THE
LARYNGOSCOPE.

VOL. LXIX APRIL, 1959 No. 4

TISSUE-CULTURE STUDIES OF OSTEOBLASTIC ACTIVITY IN OSSICULAR FRACTURES.

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Fracture of the stapedial crura is a complication seen rather frequently in present-day attempts at mobilization of the stapes. When fracture occurs, the continuity of the ossicular chain is often disrupted, and should the otologic surgeon terminate the procedure no improvement in his patient's hearing could be expected; however, some techniques employ intentional fracture of a crus, or of the footplate, to reestablish mobility of the oval window.[1,2] Also, currently, Rosen[3] advocates the creation, in the footplate, of a small hole made with a tiny metal hook, or "fenestrator," which tears through footplate bone and endosteum, allowing visible seepage of perilymph through the resulting opening. Because these methods of otologic surgery actually fracture living bone, it is of importance to study the repair process in bone following such a fracture.

For many years the impression existed that the stapes is composed of peculiar, immature bone,[4] of a type which does not heal if fractured; however, very few investigations of

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 2, 1959.

the cellular structure and pattern of ossicular bone have been carried out to see whether the bone is so unusual. Fundamentally there is the question of whether any cells possessing osteogenic potential are associated with the ossicular bone. To understand the healing process of any bone one should take a "high power" view of it, focusing attention on the cell as the primary unit for study.

The fact that ossicular fractures can and do heal by bony union has been proven by recent experimental studies. Altmann and Basek[5] state that new bone was formed in eight of 15 cases when small bone fragments of the stapedial footplate were depressed into the vestibule. Bellucci and Wolff[6] state that fracture of the stapes is usually followed by accretion of new bone regardless of site of fracture. In five cases involving fracture of stapedial crura all showed varying degrees of deposition of new bone.

Bone is often pictured as an inorganic, static structure composed of phosphate, calcium and carbonate salts. This is actually the intercellular matrix which is maintained and changed by the cellular components of this bone. Rather than a calcific conglomeration of salts it is a living, changing, metabolizing tissue composed of cells just as is all active body tissue. It is the basic unit of bone, the living cell, with which this study is concerned, especially the response of the cell to injury of the bony matrix. To make this possible tissue-culture techniques are employed.

PROCEDURE.

During the course of aseptic middle-ear operations on humans and guinea pigs the stapes was removed and placed in a vial containing sterile balanced-salt solution (Hank's). The pH of this solution was adjusted between 7.2 and 7.4 by balancing the CO_2 content. This pH was determined through observation of the color change of phenol red which was added to serve as an indicator. The balanced salt solution and the ossicles were then poured into a sterile petri dish and the bones either fractured intentionally or left intact for control purposes. Each separate stapes was then placed in a Porter flask containing a clot formed of equal parts of chicken plasma

and chicken embryo extract, which also contained the pH indicator. By occasionally opening the flask to allow the carbon dioxide to escape the pH was maintained at its original value. A few drops of horse-serum medium were placed on top of the clot and the specimen. Generally this was changed at regular intervals so as to provide a continuous supply of nutrient material. The flask was generally closed with a rubber stopper and incubated at a temperature of 38° Centigrade for various lengths of time. Cellular growth was observed microscopically, and when the activity appeared to be slowing down, the bone and clot were fixed for histological processing.

The fixative used was generally either a large quantity of 10 per cent neutral formol or Orth's fluid. In preparing the specimens for histological study a small amount of potassium dichromate, as advised by Orth,[7] was used in the fixative. This seems to harden the tissues more than plain formol, a procedure which enables the tissue to withstand the shrinking effects of higher alcohols and the heat of embedding during processing. The bones were left for about 24 hours in this solution and then decalcified for six to twelve hours in 1 per cent nitric acid. The tissues were then embedded in paraffin, sectioned at 10 microns, and after being mounted serially were stained with hematoxylin and eosin. Two human and nine guinea pig stapedes were cultured and observed by this method.

RESULTS.

In fetal life the stapes begins as a cartilage "model" which subsequently becomes ossified. This process according to Anson, Cauldwell and Bast,[8] is quite comparable to that found in a typical long bone of the body such as the tibia. A major difference, as pointed out in their article, lies in the fact that the stapes is of adult size in about 20 weeks in the fetal organism, whereas the tibia requires about 20 years to reach adult proportions. The similarity of the stapes to the long bones of the body goes even further: both have layers of cells on their surfaces, which have osteogenic ability. These cells are the so-called osteoblasts and play an important part in adding

bony substance during the growth and development stage, and also serve as the primary unit during the healing of bone fractures.

The stapedial crura are largely composed of periosteal bone which is covered by periosteal membrane and mucous membrane. Beneath the periosteal membrane is a layer of osteogenic cells recessed in jagged crevasses of the underlying

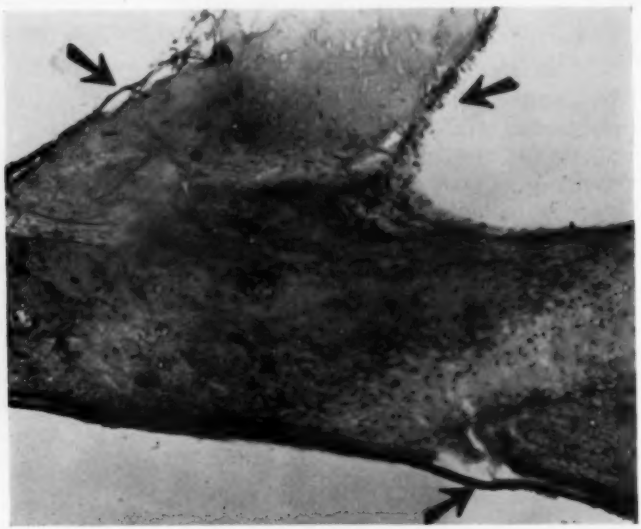

Fig. 1. Section of human stapes showing junction of anterior crus with footplate. The lower arrow points to the endosteal membrane which is continuous along footplate and inside of otic capsule. The upper two arrows point to the periosteal membrane. HTB 51R, X200.

bone. These are the cells that respond to injury when the associated bone is fractured. A similar layer of osteogenic cells is present beneath the endosteal membrane of the footplate.

Figure 1 is a photomicrograph of a sectioned normal human stapes showing these layers. This picture illustrates the crural-footplate junction and the insertion of the footplate

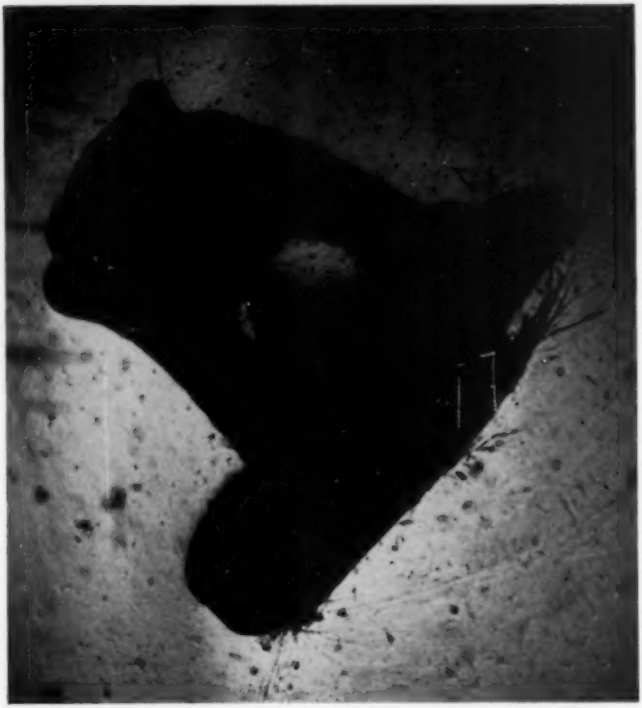

Fig. 2. Guinea-pig stapes after eight days in tissue culture. Fibroblasts can be seen growing out from site of fracture. GP 174L.

into the oval window. The periosteal bone of the crus is shown at the upper portion of the section, covered with periosteal membrane and cuboidal cells of the mucous membrane. Beneath the footplate a heavy endosteal membrane is seen which shows complete continuity to the endosteal membrane of the otic capsule. The osteogenic layer of cells lie tightly nestled in the recesses of both the periosteal and endosteal bone.

When a fractured stapes is placed in the tissue-culture medium two morphologically different types of cells are produced. Figure 2 is of a guinea-pig stapes which has had a

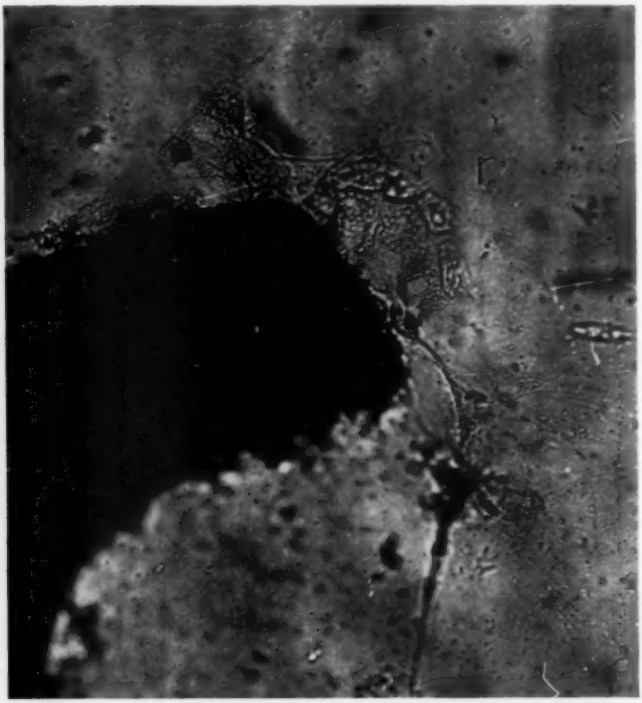

Fig. 3. Another guinea-pig stapes after eight days in tissue culture. Osteoblasts can be seen clustered around end of a fractured crus. GP 178R.

footplate fracture and then was cultured by placing it in a flask containing the chicken plasma-embryo extract clot. The picture was taken on the eighth day of culture while the specimen was still alive and growing vigorously in the Porter flask. Along the lower edge of the stapes long slender cells are proliferating and fanning out in a burst arrangement. These cells have the morphologic appearance of fibroblasts and may be arising from torn endosteal membrane.

A different type of cell is seen in Figure 3. This guinea-pig stapes was fractured and entirely separated from the footplate. The picture was taken while the bone was living and

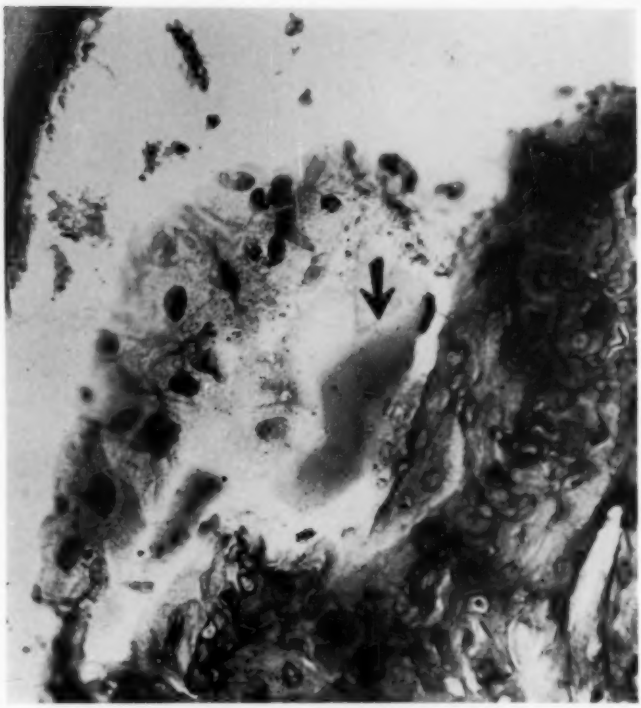

Fig. 4. Histologic section through the fracture site of stapes shown in Fig. 3. The arrow points to the osteoid tissue being laid down by the cluster of osteoblasts. Note the osteoblast being transformed into an osteocyte by becoming embedded in the osteoid material. GP 178R, X380.

after growing in tissue culture for eight days. These are plump cells with prominent nuclei and are arranged in a semicircle around the fractured end of the crus. The cells are much larger than those seen in Figure 2, and morphologically resemble osteoblasts.

The problem arises as to the nature of the cells present in these two pictures. One cannot label a cell as capable of producing a type of tissue until the cell actually has shown its ability to produce a special type of tissue. Thus an osteoblast cannot be classified with absolute certainty until it has

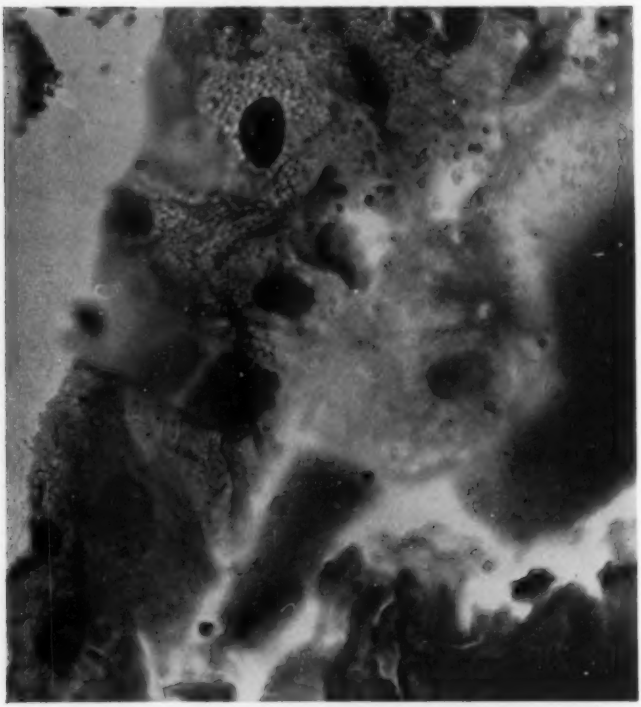

Fig. 5. Oil immersion study of small portion of area shown in Fig. 4. The typically prominent pair of nucleoli can be seen in the nuclei of several of the osteoblasts. GP 178R, X700.

aided in the production of bone. Likewise, a fibroblast cannot be definitely classified as such until it has aided in the production of fibrous tissue. In most instances, however, we must rely on the morphological and histological characteristics of the cell in question in order to classify it. It is well known that environmental factors such as pH, temperature change and the like alter these features and could result in mistaken identity if these variables were not carefully controlled.

Figure 4 is a photomicrograph of a histologic section made through the crural fracture seen in the guinea-pig stapes of

Fig. 6. A section through a second fracture site near the neck of the stapes shown in the last three figures. There is vigorous osteoblastic activity with the deposition of osteoid tissue. GP 178R, X620.

Figure 3. The upper two-thirds of the crus is depressed inward to a moderate degree and the ragged edge of bone is seen in the lower one-third portion of the crus. This stapes was in tissue culture for eight days and showed vigorous growth from the beginning. In the area of the depressed fracture a clump of osteoblasts have formed a semicircle, and are laying down a pink homogeneous material, presumably osteoid tissue. These osteoblasts probably have originated from the jagged crevasses in the periosteal bone just beneath the periosteal membrane, and have been stimulated into action by the fractured bone. These active osteoblasts resemble those decribed by Weidenreich.[9] The nucleus is large and has one or two prominent nucleoli. The cytoplasm stains with basic dyes and contains numerous threadlike mitochondria, and often one osteoblast is connected to its neighbor by thin cytoplasmic processes. The prominent nucleoli are clearly seen in Figure 4.

In Figure 5, which is a higher magnification of the area shown in the preceding figure, the cell structure is even more clearly seen. The nucleoli are especially prominent in several of the osteoblasts.

Figure 6 is a photomicrograph of a section made through another site of guinea-pig stapedial fracture. This one was made at the junction of the crura with the neck of the stapes. Mature cartilage can be seen at the lower right of the photomicrograph with dark, shrunken chondrocytes in the lucunae. This stapes, after fracture, was placed in tissue-culture media where it remained for eight days. Again we see, clustered around the site of injury, a number of active osteoblasts which were apparently engaged in laying down osteoid tissue. Some of the nucleoli can be seen in the nucleus of several osteoblasts in this figure.

The histological and morphological characteristics of these two distinct types of cells support our contention that the cells initially seen in the Porter flask in Figure 2 are fibroblasts, and those vigorously growing around a stump of a fractured crus in Figure 3 are osteoblasts. The evidence that these latter cells are capable of laying down osteoid material aids in further identifying these cells as osteoblasts. As new

bone is being formed some of the osteoblasts become embedded with this osteoid matrix and are then known as osteocytes.[10] Their function there is to maintain and nourish the surrounding bony matrix. Shown in Figures 4 and 5 is an osteoblast becoming enmeshed in this osteoid tissue, apparently beginning its transformation into an osteocyte.

The bony tissue shown in the above figures was being formed by cells of the periosteal layer and thus is produced by intramembranous ossification. This is the same manner in which new bone is added to the shaft of a mature long bone of the body. Such intramembranous formation of bone was the only type observed in the repair of stapedial fractures in tissue-culture study.

Histologically, guinea-pig stapedes appear practically identical to those of humans; however, when the human stapedes were fractured and placed in a tissue-culture medium the fracture site produced heavy growth of fibroblasts and osteoblasts but no evidence of osteoid tissue formation. There are several factors which may account for this.

Not all presumably living bone when removed from a living animal shows cellular outgrowth when placed in the culture medium, and the production of osteoid tissue is even rarer. Only two of the nine guinea-pig bones showed this type of tissue formation, although extensive osteoblastic activity was seen in the others, and it may have been that the use of only two human stapedes was not a large enough sample. Failure of the human material to produce osteoid tissue might also be attributed to the technique for obtaining and transporting the bone from operating room to laboratory. This necessitated a longer time stay for the bits of bone in the balanced-salt solution at variable temperatures before being placed in the culture medium and incubator.

It is also possible, of course, that human-stapedial bone does not react in tissue culture to the stimulus of a fracture, as does guinea-pig stapedial bone; however, the striking evidence of their close morphologic and histologic response in tissue-culture medium tends to support the impression that they are very similar to one another. Both produced extensive

outgrowth of fibroblasts when the mucous and periosteal membranes were traumatized, and both showed evidence of osteogenic potential by production of osteoblasts at or near the site of fracture; therefore, it seems highly probable that the failure of the human material to produce osteoid tissue was due either to technique or chance, and that in the living organism healing should take place in most stapedial fractures whether the host organism is human or animal.

COMMENT.

Unquestionably an osteogenic layer of dormant osteoblasts does exist along the surface of ossicular bone. These osteoblasts respond to a stimulus such as fracture and carry out a repair process similar to that found in bone elsewhere in the body. Ossicular bone is a living, cellular tissue whose size, shape and nutrition depend on living cells, or osteocytes. Although these studies were carried out by tissue-culture methods we have no reason to believe that these osteoblasts and fibroblasts should react differently in a living human organism. Human cancellous bone frozen for several days has shown production of osteoid tissue when placed in tissue culture by Ray and co-workers[11]; therefore, there is no reason to suppose that fracture of the crura or footplate would remain in a state of non-union. Any fracture that is made will have a tendency to heal resulting in return of the patient's hearing to the pre-operative level; also, the extensive production of fibroblasts indicates that fibrosis can result from middle-ear manipulations.

SUMMARY.

Through the method of placing fractured stapes in tissue-culture media and observing the cellular activity both grossly while the tissue is living, and microscopically after histologic processing, it has been demonstrated that:

1. Injury to the periosteal membrane results in fibroblastic proliferation;

2. Tearing of the osteogenic layers, together with bone fracture such as occurs in footplate fenestration or crural

fracture, stimulates new bone formation with a gloomy prognosis for either keeping this "fenestra" open, or for preserving the fracture separation.

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NEW RESEARCH LABORATORY.

The Elbyrne G. Gill Eye and Ear Foundation, 711 So. Jefferson St., Roanoke, Va., announces the establishment of a Research Laboratory in conjunction with the Eye Bank and Sight Conservation Society of Virginia under the direction of Miss Jean Swartz, M.S. in chemistry. The laboratory is prepared to make the dye test for Toxoplasmosis.

MANAGEMENT OF CARCINOMA OF THE LARYNX AND LARYNGO-PHARYNX.*

Recent Experiences.

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The treatment of malignant disease of the larynx and of the laryngo-pharynx is a controversial matter of world-wide interest. In the past, the rival and often conflicting claims of surgical excision and of radiotherapy have been hotly debated without reaching any definite conclusions, but in Britain a measure of agreement is gradually emerging.

This has been brought about by a better knowledge of what can, and what cannot, be achieved by these differing forms of therapy. Closer cooperation between surgeon and radiotherapist has done a great deal to bring about this understanding. Combined clinics have been established in which every patient is seen by an experienced expert in each field; thus, a patient attending a joint consultation clinic is assured of a carefully considered opinion as to the method of treatment most likely to be of benefit. Each new case is an individual problem, and treatment is advised in the light of the anatomical situation and extent of the lesion, the pathology, the facilities available for treatment and the particular circumstances and wishes of each patient. The results of treatment are followed up regularly by both members of the team, and secondary treatment for recurrence is instituted with a greater degree of certainty at the earliest possible moment. Such a clinic was established at the Metropolitan Ear, Nose and Throat Hospital, and I have had the opportunity and ex-

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*Read by invitation at the Meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, New York, N. Y., Jan. 8, 1959.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 19, 1959.

perience of working in close collaboration with my colleague, Dr. M. Lederman, of the Royal Marsden Hospital, an institute which specializes in the treatment of malignant disease, and which is well equipped with modern radiotherapeutic apparatus.

Recent advances in the technique of radiotherapy and of surgery have been considerable. I am not competent to deal with the former, but have acquired some experience in the

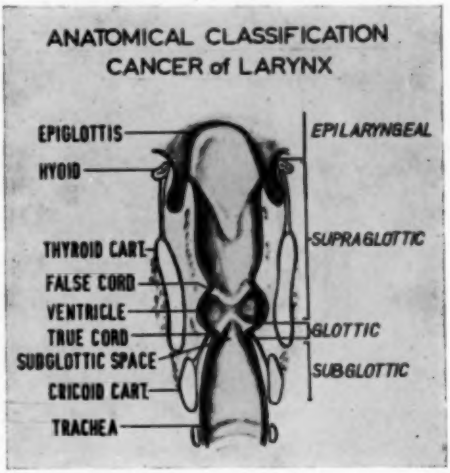

Fig. 1. Anatomical classification of cancer of the larynx. An imaginary line through the hyoid bone divides the epiglottis into two: Tumors arising above this line are classified with laryngo-pharyngeal carcinomata.

newer operative methods. Better and safer anesthesia, and greater use of blood transfusion and of antibiotics have allowed the performance of major *en bloc* resections without undue risks and without an unduly high operative mortality.

Surgical excision has thus been extended to include widespread neoplasms formerly considered to be inoperable. On the other hand, the trend has been to treat by radiotherapy alone early localized lesions, with surgery reserved for the failures. There has thus been a reversal of the treatment

plan, which was formerly adopted, whereby early tumors were removed and advanced neoplasms were given radiotherapy. In consequence, the radiotherapist is having the first chance of treating the early cases which are more likely to be curable, a most important fact which must be borne in mind constantly when comparing the results of therapy.

A system of classification into anatomical types, clinical

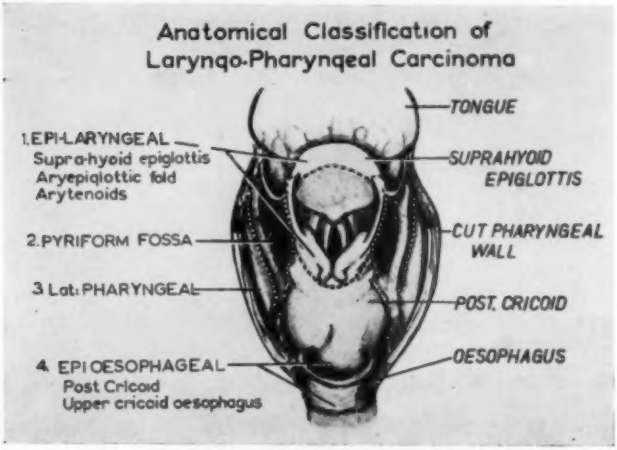

Fig. 2. Anatomical classification of laryngo-pharyngeal cancer. Tumors arising from the anterior surface of the epiglottis, the valleculae and the posterior part of the tongue are ecluded—they are glosso-epiglottic eoplasms.

stages and histological groupings is vitally necessary to make for a terminology common to radiotherapists and to laryngologists, in order to compare like cases with like in any statistical review. In the past, comparisons between the two forms of treatment have been misleading and unscientific, as widely differing criteria have been adopted in the publication of results.

It is, therefore, opportune to take stock of the present position against this background.

PERSONAL MATERIAL.

Individual British otolaryngologists are unable to collect as large a series of cases as their American colleagues. In Britain, the otolaryngologist usually remains more general in his outlook, and there is little ultra-specialization within the province of ear, nose and throat work; thus, the available material is distributed among a larger number of specialists. This fact, coupled with the invasion of the general surgeon into our field, reduces the numbers seen by British specialists as compared with American laryngologists who tend to concentrate their endeavors on a particular disease within the

TABLE I.

ANATOMICAL DISTRIBUTION—108 CASES.

	Male	Female	All
Cancer of Larynx (64 Cases)—			
Supraglottic	6	1	7
Glottic	40	3	43
Subglottic	12	2	14
Cancer of Laryngo-Pharynx (44 Cases)—			
Epilaryngeal	10	0	10
Sinus Pyriformis	7	1	8
Epiesophageal	6	20	26
Total (108 Cases)	81	27	108

specialty. This introduction is necessary to explain the small number of personal cases of carcinoma of the larynx and of the laryngo-pharynx which form the basis of this report—one concerned with individual experience.

This paper deals with a series of 108 cases (see Table I), which have been under my own care in the last nine years. This approach has been deliberately chosen in preference to one relating to a large number which have been culled from case-notes borrowed from many sources and concerning patients who have been treated by several surgeons in the same hospital. It allows a considered presentation of personal opinions gained solely by *first hand* experience in the management of this disease. The time limitation is also deliberate. Earlier cases have been excluded for several reasons:

1. Relative inexperience at first.

2. Deficiencies in case-taking, record-taking and follow-up, making classification and an accurate report impossible.

3. The remarkable improvement in selection of cases for treatment in the last decade.

4. The recent advances in radiotherapeutic and surgical techniques which bring into focus one's own changing views by concentrating on current methods of therapy.

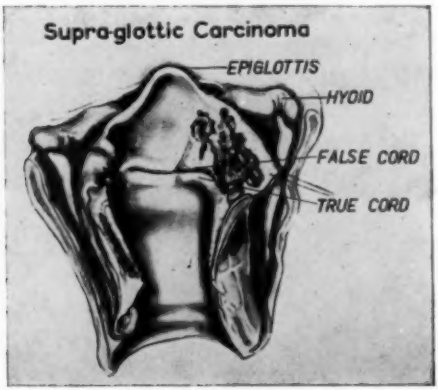

Fig. 3. Supraglottic type of cancer of the larynx. Artist's impression of a laryngectomy specimen, the tumor arising from the infra-hyoid epiglottis and false cord.

CLASSIFICATION OF CASES.

1. Anatomical Site.

For many years carcinoma of the larynx was divided clinically into an intrinsic and an extrinsic group. In view of the vastly different prognosis in these two types, such a broad distinction is inadequate. An anatomical classification according to a more exact site of origin, as suggested by Lederman[1,2] (1952-1954), has been adopted.

A. Cancer of the Larynx is divided into:

1. Supraglottic: Tumors arising from Infra-Hyoid Epiglottis; False Cord; Ventricle.

2. Glottic: Tumors arising from upper surface and edge of true vocal cord; anterior and posterior commissures.

3. Subglottic: Tumors arising from: inferior surface of cord; subglottic space.

B. Cancer of the Laryngo-Pharynx is divided into:[2]

1. Epilaryngeal: Tumors arising from supra-hyoid epiglottis; aryepiglottic fold and arytenoid.

2. Sinus pyriformis.

3. Epiesophageal — Cricopharyngeal; pharyngo-esophageal; cervical esophagus.

4. Posterior and lateral pharyngeal walls.[2]

The distribution of cases in this series according to site and sex is shown in Table I.

2. Clinical Staging.

The prognosis is related to the extent of invasion which is present when treatment is undertaken. The system of clinical grading followed at the Metropolitan Ear, Nose and Throat Hospital is as follows (Lederman, M.[1,2]):

A. Laryngeal Cancer: Stage

1. The tumor is limited to the tissue of origin and the larynx is freely mobile;

2. Laryngeal mobility is impaired or extension has occurred beyond the tissues of origin but still remains within the confines of the larynx.

3. The affected part of larynx is fixed or extra-laryngeal spread is present to thyroid cartilage, pre-epiglottic space, trachea, laryngo-pharynx or to a single mobile homolateral lymph node.

4. Contra-lateral, bilateral or fixed lymph nodes, simultaneous or consecutive primary or distant metastases are present.

B. Laryngo-pharyngeal Cancer: Stage

1. Limited to tissue or origin.

2. Local spread but still within the confines of the pharynx or of the cervical esophagus.

3. Spread outside walls of pharynx or cervical esophagus.

4. Contra-lateral, bilateral or fixed lymph nodes, distant metastases or simultaneous or consecutive primary cancer.

Table II shows the number of patients and distribution according to site and stage.

TABLE II.

DISTRIBUTION OF CASES ACCORDING TO SITE AND STAGE.

	Stage 1	Stage 2	Stage 3	Stage 4
Supraglottic (7 Cases)	0	2	4	1
Glottic (43 Cases)	14	11	17	1
Subglottic (14 Cases)	1	1	11	1
Epilaryngeal (10 Cases)	0	1	7	2
Sinus Pyriformis (8 Cases)	0	0	3	5
Epiesophageal (26 Cases)	0	2	16	8
Total (108 Cases)	15	17	58	18

3. Histological Grouping.

The allocation of a particular biopsy specimen to one of the four histological groups suggested by Broders[3] (1926), is misleading and has been discontinued. The small portion submitted for biopsy may be unrepresentative of the tumor as a whole, and pathologists differ in their groupings of the intermediate varieties, *i.e.*, those between the well differentiated and the anaplastic types (Colledge)[4].

Precise division into well differentiated, intermediate and anaplastic groups is possible only when the whole tumor is available for examination. Surgically excised neoplasms can be divided into these three groups, but when assessing the grouping on a small biopsy specimen, inaccuracy is avoided by taking a broader classification into frank squamous carcinoma, anaplastic carcinoma and recurrent squamous carcinoma. The histological classification in this series, according to this basic grouping, is shown in Table III.

In this analysis, cases of *in situ* carcinoma and of borderline malignant lesions are excluded. Herein lies one of the main sources of error in histological interpretation. What one pathologist reports as negative, another will regard as probably malignant. Their inclusion helps to improve the statistical cure-rate, but confuses the real issue, the true prognosis. For who is to say how many of these are false positives? An unduly optimistic picture of the disease emerges when such questionable lesions are included with frankly malignant tumors.

The bad prognosis associated with immature, anaplastic lesions is well known. It is, therefore, most important to recog-

TABLE III.

HISTOLOGICAL GROUPING. 108 CASES.

	Frank Squamous Carcinoma. Primary or Recurrent	Anaplastic Carcinoma
Supraglottic	7	0
Glottic	39	4
Subglottic	11	3
Epilaryngeal	9	1
Sinus Pyriformis	8	0
Epiesophageal	26	0
	100	8

nize this group as a separate entity. They numbered eight in this series, seven of these occurring in the glottic and subglottic regions.

BIOPSY.

Direct laryngoscopy and biopsy give a certainty of diagnosis that can be obtained in no other way. This essential investigation is never delegated to an assistant, but is personally performed. The exact position and probable extent of the growth is ascertained, and a piece is removed from the most likely portion of the tumor. The percentage of false negative reports is thereby reduced, but absolute reliance is not placed on negative biopsy findings alone. Two false negative biopsies were returned in one of my cases, but despite this, laryngectomy was performed on clinical grounds. The clinical diag-

nosis was confirmed subsequently by a positive report when the whole specimen was searched histologically. This error on biopsy is especially liable to occur with subglottic growths.

TREATMENT AND RESULTS.

Part I. Cancer of the Larynx.

Treatment has been related to the site, stage and to a lesser degree to the type of cell comprising the tumor.

In general, anaplastic neoplasms in all areas are not subjected to primary surgery unless the lesions are early and well

TABLE IV.

TREATMENT AND RESULTS—SUPRAGLOTTIC LESIONS.

Case No.	Sex	Age	Clinical Stage	Treatment	Result
1.	F	62	4	T-R only	Died with disease in 3/12.
2.	M	62	3	T-R-LY	Alive. Free of disease in 40/12.
3.	M	59	2	T-R-LY	Alive. Free of disease in 22/12.
4.	M	74	2	T-R-LY	Alive. Free of disease in 16/12.
5.	M	73	3	T-C	Alive with disease in 12/12.
6.	M	55	3	LY-D.X.R. Post op.	Died with disease in 9/12.
7.	M	69	3	D.X.R. only	Died with disease in 5/12.

Abbreviations: T-R—Teleradium; LY—Laryngectomy; T-C—Telecobalt; D.X.R.—Deep X-ray Therapy; –/12 Time in months.

localized. Laryngofissure is unsuccessful in Stage 1 anaplastic cases and mutilating radical operations alone have less chance of controlling the disease. Primary radiotherapy is given in such cases, and surgery is reserved for recurrence. An *en bloc* dissection is advisable in conjunction with radical surgery whenever there is a likelihood of clinical involvement of glands, as secondary neck dissection has less chance of affecting a cure.

In non-anaplastic squamous carcinomata, treatment is selected according to the clinical stage and site of the neoplasm, and various combinations of therapy have been tried. It is now usual to treat early glottic disease by radiotherapy, reserving surgery for advanced cases, uncontrolled primary lesions, recurrence after radiotherapy, and for cervical glandular metastases.

Supra-Glottic Lesions. Primary treatment by radiotherapy followed by laryngectomy has given the best results (see Table IV). Metastases to glands occur infrequently and do not warrant "white" *en bloc* neck dissection; but when nodes are manifest this operation is performed in preference to radiotherapy, either contemporaneously with laryngectomy or later, when clinical involvement occurs postoperatively.

Fig. 4. Glottic type of cancer of the larynx. A laryngectomy specimen photographed to show massive growth involving the whole length of the right true cord as seen from behind. The section of tumor removed postoperatively for thorough histological study is well demonstrated at the mid-point of the cancer.

Glottic Disease. This is the most frequent type of lesion, but the total number of cases in this series is too small to make a worthwhile and accurate analysis of the percentage of five-year cures attained by surgery, by radiotherapy or by their combination. Instead, a detailed analysis of the outcome of treatment of each case is studied. This searching inquisition is used with the object of discovering the treatment most likely to achieve success in the future.

At the outset, Stage 1 and Stage 2 cancers were usually treated by laryngo-fissure or by laryngectomy, respectively,

but in view of the success obtained with teleradium and telecobalt therapy, with preservation of full laryngeal function, primary surgery was largely abandoned in favor of radiotherapy. With Stage 3 disease the number of cases requiring secondary laryngectomy was high, especially when there was unilateral involvement of glands; then, primary laryngectomy, combined with *en bloc* dissection, was the usual method of treatment.

Table V shows the primary treatment adopted in the 43

TABLE V.

PRIMARY TREATMENT IN 43 GLOTTIC CASES.

Clinical Stage	Radiotherapy (19 Cases)	Laryngofissure (14 Cases)	Laryngectomy (9 Cases)	Palliative (1 Case)
1	3	14	0	0
2	6	0	2	0
3	10	0	7	0
4	0	0	0	1

cases falling within the glottic group and Tables VI, VII, VIII, IX show their fate.

Primary Radiotherapy.

Of the 19 primarily irradiated cases, seven required laryngectomy for recurrence, all of these being clinical Stage 3, and of these seven, five died of the disease. Stage 1 and 2 disease does well with radiotherapy, but laryngectomy is often necessary for more advanced lesions.

Primary Thyrotomy.

The primary laryngo-fissure (thyrotomy) cases (all Stage 1) are analyzed in Table VII. From this it will be seen that seven are free of disease, but seven required secondary laryngectomy. All the laryngectomized patients have done well, six being alive and free from disease for periods ranging from nine years to six months, while the seventh patient died from intercurrent illness but free from malignant disease (proved by postmortem) three-and-a-half years after laryngectomy.

Two of the seven were of anaplastic histology, and today

would not be treated by laryngo-fissure. Luckily, both of these patients are free from disease nine years after laryngectomy. One patient developed a squamous cell carcinoma on the remaining cord six years after laryngo-fissure, and this is probably a second primary rather than a recurrence, as no carcinoma was found on the operated side. Two of the other patients had leukoplakia of larynx and subsequently developed

TABLE VI.

GLOTTIC CARCINOMA—ANALYSIS OF 19 PRIMARY RADIOTHERAPY CASES.

No.	Sex	Age	Clinical Stage	Histology	Form of R-Thy.	Sec. Treat. Recur.	Fate
1.	M	54	2	Sq.	T-R	—	A>5 years
2.	M	33	1	Sq.	T-R	—	A>5 years
3.		58	1	Sq.	T-R	—	A>4 years
4.	M	62	2	Sq.	T-R	—	A>4 years
5.	M	70	2	Sq.	T-R	—	A>3 years
6.	M	53	3	Sq.	D.X.R. T.C.	LY for Perichondritis	D (Post-op.)
7.	F	74	3	Sq.	T-R	—	D 3/12
8.	M	76	2	Sq.	T-R	—	A>2 years
9.	M	72	3	Sq.	T-R	LY	A>4 years
10.	M	51	3	Sq.	T-R	—	A>2 years
11.	M	61	1	Sq.	T-R	—	R>2 years (2ndries Ribs)
12.	M	69	2	Sq.	T-R	—	D 10/12
13.	M	64	3	An.	T-R	LY, 2° Block, H.V.T.	D 6/12
14.	M	49	3	Sq.	T-R	LY	D 14/12
15.	M	62	2	Sq.	T-C	—	A 12/12
16.	M	69	3	Sq.	T-R	LY	D 15/12
17.	M	71	3	An.	T-R	LY	A>4 years
18.	F	67	3	Sq.	T-R	—	A 18/12
19.	M	46	3	Sq.	H.V.T.	LY	D 11/12

Key: F—Female; M—Male; Sq.—Squamous; An.—Anaplastic; T-R—Teleradium; T-C—Telecobalt; H.V.T.—High Voltage Therapy; LY—Laryngectomy; A—Symptom free; R—Alive with disease; D—Died of disease; >—Longer than.

recurrent carcinoma on the opposite side. Today such cases would not be treated by laryngo-fissure, in view of the premalignant nature and the likelihood of multicentric malignant degeneration. The remaining two patients were found to have histological evidence of malignant disease on the opposite cord, although there was no clinical evidence of this at the time of the laryngo-fissure. A routine biopsy is always taken from the junction of the anterior and middle thirds of the

apparently healthy cord, or from any other suspicious area. These two cases substantiate the wisdom of this, as both patients were submitted to laryngectomy within a short period of the primary operation and have remained free of disease. Thus, cancerous tissue is widely excised at the earliest possible moment by performing an adequate operation.

Incidentally, the thyroid ala on the affected side is also removed and examined histologically. Invasion was not found

TABLE VII.

GLOTTIC CARCINOMA.—ANALYSIS OF 14 LARYNGOFISSURE CASES—ALL CLINICAL STAGE 1.

No.	Sex	Age	Histology	Result of Fissure	Sec. Treat.	Final Result after LY
1.	M	66	Sq.	Recurrence 6 years (? 2nd Primary)	LY	A>3 years
2.	M	65	An.	Recurrence	LY	A 9 years
3.	M	43	An.	Recurrence	LY and 1^{r} Block	A 9 years
4.	M	74	Sq.	Recurrence	LY	I.D. 32/12
5.	M	60	Sq.	Recurrence	LY	A>5 years
6.	M	64	Sq.	+ Biopsy opp. cord.	LY	A 2½ years
7.	M	62	Sq.	A 5 years	—	—
8.	M	78	Sq.	A 18/12	—	—
9.	M	60	Sq.	A 8 years	—	—
10.	M	62	Sq.	A 5 years	—	—
11.	M	57	Sq.	A 7 years	—	—
12.	M	65	Sq.	+ or biopsy opp. cord.	LY	A 6/12
13.	M	65	Sq.	A 5 years	—	—
14.	M	62	Sq.	A 4 years	—	—

Key: as before. Added: I.D.—died intercurrent disease free from malignancy.

in these cases, but had this occurred, an early secondary laryngectomy would have been performed. Histological studies such as these do sometimes prove the preoperative clinical staging to be incorrect.

Primary Laryngectomy.

Table VIII summarizes the nine primary largnectomy cases individually.

Three developed recurrence; of the remaining six, two patients died of intercurrent disease but proved to be free

from recurrence at postmortem, and the remaining four have remained free from disease for periods between eight years and over two years. A comparison of the end-results of the two primary methods in Stage 3 disease shows that four out of ten primarily irradiated patients are symptom free, but three of these survivors required secondary laryngectomy, whereas four out of seven of those treated by primary surgery remained symptom free; however, five out of seven

TABLE VIII.

GLOTTIC CARCINOMA—ANALYSIS OF 9 PRIMARY LARYNGECTOMY CASES.

No.	Sex	Age	Clinical Stage	Histol.	Prim. Treat.	Result Prim. Treat.	Sec. Treat.	Final Result
1.	M	56	3	Sq.	LY	Glands in 2/12	Block Diss. 2/12	D 18/12
2.	M	35	3	Sq.	LY	A 8 years	—	—
3.	M	73	2	Sq.	LY	I.D. 42/12	—	—
4.	M	68	3	Sq.	LY + Prim. Block	Recur. Esoph. 4 years	Resection Esoph. + Block, Contra. Lat.	R 56/12
5.	M	69	3	Sq.	LY	I.D. 2/12	—	P.M. No Ca.
6.	M	58	3	Sq.	LY	A>2 years	—	—
7.	M	70	2	Sq.	LY	A>2 years	—	—
8.	M	72	3	Sq.	LY	A>7 years	—	—
9.	M	67	3	Sq.	LY	Recur. glands 2 years	Block Diss. 2 years	D 4 years

patients (Stages 2 and 3) primarily irradiated and secondarily laryngectomized died of the disease (see Table IX).

In the light of my present experience, radiotherapy, therefore, seems to be the primary treatment of choice for clinical Stages 1 and 2, with secondary laryngectomy reserved for uncontrolled disease, recurrence or anaplastic tumors. With clinical Stage 3 disease, primary laryngectomy offers the patient the best chance of cure; but anaplastic cases are irradiated first, and neck dissection is subsequently combined with secondary laryngectomy. This present analysis bears out my clinical impressions previously expressed elsewhere,[5] and the opinions of other authors: McCall and Fisher[6] (1952), Schall[7] (1951), Gissillson and Lindgren[8] (1952), Lindsay and Ironside[9] (1955), etc.

Subglottic Lesions. The treatment of choice for subglottic carcinoma is regarded as surgical by most laryngologists, laryngectomy and block dissection giving the best results (Orton,[10] 1951; Ogura,[11] 1955; Work,[12] 1952).

Radiotherapists have achieved some successes, however, especially when the lesion is early (Lederman,[1] 1952). In this series, six out of 14 cases were treated by primary radiotherapy (see Table X). From this it will be seen that of those

TABLE IX.

GLOTTIC CARCINOMA—RECURRENT OR UNCONTROLLED—SECONDARY LARYNGECTOMY CASES (14).

No.	Prim. Treat.	Prim. Clin. Stage	Subsequent Stage After Recurrence	Original Histol. Group	Result = Time After LY
1.	L-F	1	2	Sq.	A 3 years
2.	L-F	1	3	An.	A 9 years
3.	L-F	1	3	An.	A 9 years
4.	L-F	1	2	Sq.	I.D. 32/12
5.	L-F	1	3	Sq.	A 5 years
6.	L-F	1	2	Sq.	A 2½ years
7.	L-F	1	2	Sq.	A<1 year
8.	T-R	3	3	Sq.	A 4 years
9.	T-R	2	3	Sq.	D 15/12
10.	H.V.T.	3	3	Sq.	D 11/12
11.	T-R	3	3	An.	A 4 years
12.	T-R	3	3	An.	D 6/12
13.	T-R	3	3	Sq.	D 14/12
14.	T-R	3	3	Sq.	D 15/12

six, four were subjected to secondary laryngectomy. The operation was offered to the other two, but was declined, and it is interesting to note that the only Stage 1 patient who had anaplastic disease, and who was treated solely by irradiation, has remained free of disease for over five years and has normal vocal function. The two other patients with anaplastic histology have died of recurrence. Only one of these received preliminary radiotherapy, and looking back it would have been advisable to have given this treatment prior to surgery in the other. Postoperative radiotherapy will not cure the disease if surgery fails, but is used to palliate the condition.

Of the eight cases treated by primary laryngectomy, five are alive and free of disease, but only two of these have

reached the five-year period; two are dead, and one is alive with uncontrolled malignancy.

Only one patient had clinically involved glands (bilateral) before treatment was started, necessitating a bilateral monoblock resection of gland fields and larynx; he has remained well for 17 months. Prophylactic neck dissection is not performed routinely in this group because of the relatively low incidence of involvement of the cervical nodes (13 per cent, according to Lederman[13]).

TABLE X.
SUBGLOTTIC CARCINOMA—ANALYSIS OF 14 CASES.

No.	Sex	Age	Clinical Stage	Histol.	Prim. Treat.	Secy. Treat.	Result
1.	F	39	1	An.	T-R	Nil	A 5 years
2.	M	76	2	Sq.	T-R	H.V.T.	R 3½ years
3.	M	58	3	Sq.	LY	—	A 8 years
4.	M	71	3	Sq.	LY	—	A 5 years
5.	M	53	3	Sq.	LY	H.V.T.	D 5/12
6.	M	63	3	An.	LY	—	D 1 year
7.	M	56	3	Sq.	T-R	LY	D 3/12
8.	M	64	3	Sq.	H.V.T.	LY	I.D. 14/12
9.	M	59	3	Sq.	T-R	LY	A 1 year
10.	M	48	3	An.	T-R	LY	D 7/12
11.	M	62	3	Sq.	LY	—	A 14/12
12.	M	71	3	Sq.	LY	—	A 2 years
13.	F	59	3	Sq.	Hemi-LY + T-R	H.V.T.	R 14/12
14.	M	52	4	Sq.	LY + Bilat. Block	—	A 17/12

In the future, it is intended to use radiotherapy as a preliminary to surgery in anaplastic disease, and to employ it as the first choice of treatment for the rare, early Stage 1 cases. All others will continue to be treated by primary laryngectomy, with a combined monoblock neck dissection when the glands are clinically involved.

Part II. Cancer of the Laryngo-Pharynx.

The poor prognosis associated with surgical treatment of this group of cancers in the past led to the custom of treating them by radiotherapy. Disappointed with the results of radiotherapy, laryngologists are again veering towards surgery now that very radical excision is possible. This is a world-

wide trend, and it is opportune to inquire whether our present day results justify extensive and mutilating operations. It is no wonder that some patients decline operation when the position is fully explained to them, but it is only right that this should be done; thus, the treatment is guided, to some extent, by the patient's own wishes. When surgery is undertaken, excision implies a wide-field, monoblock removal of the tumor and of the lymphatic channels and gland fields on one

TABLE XI.

EPILARYNGEAL CARCINOMA (10 CASES).

No.	Sex	Age	Clinical Stage	Histol.	Prim. Treat.	Result Prim. Treat.	Sec. Treat.	Result
1.	M	53	3	Sq.	Reg. Excision	No recurrence	H.V.T.	A>6 years
2.	M	54	3	Sq.	Reg. Excision	Osteomyelitis mandible 5 years later	H.V.T.	A>5 years
3.	M	63	3	Sq.	T-R	Uncontrolled 1^y	Reg. Excision	D 23/12
4.	M	64	3	An.	H.V.T.	Uncontrolled 1^y	Reg. Excision	D 16/12
5.	M	57	3	Sq.	H.V.T.	Uncontrolled 1^y	Refused Op.	D 3/12
6.	M	82	2	Sq.	Pall. H.V.T.	Died during H.V.T.	—	D 10 days
7.	M	36	4	Sq.	(Incomplete) H.V.T.	Uncontrolled $1^y + 2^y$	—	D 4/12
8.	M	55	2	Sq.	Reg. Excision	Local Recurrence	—	D 10/12
9.	M	60	3	Sq.	Reg. Excision	Pulmonary Metastases	—	D 10/12
10.	M	64	2	Sq.	H.V.T. (Incomplete)	Died during H.V.T.	—	D 15 days

or both sides, because of the likelihood of manifest or occult lymphatic spread. Regional surgery, rather than local excision is, therefore, indicated.

Epilaryngeal Lesions. There are ten epilaryngeal cancers which are individually analyzed in Table XI. The poor results obtained by various combinations of regional surgery and radiotherapy are disappointing. There are only two survivors free of disease after a period of over five years, treated by regional surgery and postoperative irradiation; one of these survived five years, but developed a secondary osteo-

myelitis of the mandible recently. In this small number of cases this treatment plan appears to give the best results, as there are no survivors treated by primary radiotherapy.

Sinus Pyriformis Lesions. In view of the poor prognosis, seven patients in this group were treated primarily by radiotherapy and one was palliated; only one was subjected to secondary regional surgery, which proved unsuccessful (see Table XII).

TABLE XII.
SINUS PYRIFORMIS CARCINOMA (8 CASES).

No.	Sex	Age	Clinical Stage	Histol.	Prim. Treat.	Result Prim. Treat.	Sec. Treat.	Final Result
1.	M	59	4	Sq.	H.V.T.	Uncontrolled	—	D 11/12
2.	M	75	3	Sq.	T-R	Controlled		I.D. 3 yrs. 9/12
3.	M	81	3	Sq.	T-R	Uncontrolled		D 1 year
4.	M	45	4	Sq.	H.V.T.	Uncontrolled	—	D 10/12
5.	M	74	4	Sq.	T-R	Uncontrolled	Reg. Excision + Post. op. T-R	D 9/12
6.	F	72	3	Sq.	(T-C Incomplete)	Uncontrolled	—	D 14 days
7.	M	51	4	Sq.	T-C	Uncontrolled	—	R 7/12
8.	M	63	4	Sq.	Nil	Palliated	—	D 2/12

The great majority of the pyriform sinus neoplasms are very advanced before treatment is begun. Perhaps the British cases are seen much later than those in the U. S. A., a fact which would account for the wide differences in the percentage cure rates reported by British and American authors. The reported results obtained by adequate surgical removal are superior to those achieved by radiation, and in suitable early cases regional surgery is advisable. To date, all cases seen by me have been too advanced to warrant primary surgical intervention. Irradiation has only been of palliative help, and in no instance has it controlled the malignancy in the seven cases so treated.

Epiesophageal Lesions. These 26 patients also present a sorry picture, for no patient has survived longer than 13 months, irrespective of the type of treatment adopted (see Table XIII). Many cases were very advanced when first seen,

TABLE XIII.

EPIESOPHAGEAL CARCINOMA (26 CASES).

No.	Sex	Age	Clinical Stage	Histol.	Prim. Treat.	Result 1° Treat.	Sec. Treat.	Final Result
1.	F	56	4	Sq.	Reg. Excision (Incomplete)	Uncontrolled Incomplete Removal	Too advanced	D 4/12
2.	M	62	4	Sq.	H.V.T. (Incomplete)	Uncontrolled	—	D 12 days
3.	F	68	3	Sq.	T-R (Incomplete)	Uncontrolled	—	D 8 days
4.	F	41	3	Sq.	H.V.T. (Incomplete)	Uncontrolled	—	D 2/12
5.	M	74	3	Sq.	None	—	—	D 9 days before any treat. given.
6.	M	82	3	Sq.	H.V.T.	Uncontrolled	—	D 10/12
7.	F	80	2	Sq.	T-R	Uncontrolled	H.V.T.	D 13/12
8.	M	63	4	Sq.	H.V.T.	Uncontrolled	Reg. Excision (Incomplete)	D 11/12
9.	F	56	2	Sq.	T-R	Uncontrolled	—	D 4/12
10.	F	75	3	Sq.	T-C (Incomplete)	Uncontrolled	—	D 20 days
11.	F	50	3	Sq.	Reg. Excision	No Ca. at P.M.	—	I.D. 8/12
12.	F	57	4	Sq.	Reg. Excision (Incomplete)	Sub-Clavicular glands involved. Not removed.	—	R 10/12
13.	F	63	3	Sq.	Reg. Excision	Postmortem No recurrence	—	I.D. 8/12
14.	F	58	4	Sq.	Surgical Exploration Inoperable	Uncontrolled	—	D 9/12
15.	F	54	3	Sq.	Reg. Excision	Uncontrolled	—	D 9/12
16.	F	46	3	Sq.	Reg. Excision	Uncontrolled	—	D 7/12
17.	M	79	4	Sq.	Reg. Excision (Incomplete)	Uncontrolled	—	D 5 days Post. op.
18.	F	64	3	Sq.	Reg. Excision	—	—	D 1 day Post. op.
19.	F	64	4	Sq.	Reg. Excision	—	—	D 4 hours Post. op.
20.	F	54	4	Sq.	Reg. Excision (Incomplete)	Died on Table	—	D at operation
21.	F	61	4	Sq.	Reg. Excision (Incomplete)	Uncontrolled	—	D 2/12
22.	F	68	3	Sq.	Reg. Excision	Uncontrolled	—	D 8/12
23.	F	69	3	Sq.	Reg. Excision	Uncontrolled	—	D 6/12
24.	M	63	4	Sq.	Reg. Excision	Uncontrolled	—	D 5/12
25.	F	54	3	Sq.	Reg. Excision	Uncontrolled	—	D 3/12
26.	F	54	4	Sq.	T-C (Incomplete)	Uncontrolled	—	D 7 weeks

and some were in no fit state to withstand major surgery, or even radiotherapy. The anatomical site of the growth was primarily in the cervical esophagus in male patients with secondary spread upwards, whereas in females it was in the hypopharynx or in the junctional region between hypopharynx and esophagus, making an accurate anatomical classification into hypopharyngeal and cervical esophageal types impossible; hence, all cases have been classified as epiesophageal. This broad classification does not seem to matter, from a general point of view, as all cases have the same unfavorable outcome; but from the practical one, successful surgical excision is unlikely when the greater part of the upper esophagus is invaded.

TABLE XIV.
TREATMENT ADOPTED FOR EPIESOPHAGEAL LESIONS.

	Prim. Regional Excision (15)	Primary Radio-Therapy (9)	Surgical Exploration (Inoperable)	Palliative Treatment only	Secondary Reg. Exc. IY R-Thy.
Curative (Treat. completed)	10	4	1	..	..
Palliative (Treat. incompleted)	5	5	..	..	1
Totals	15	9	1	..	1

The surgical treatment adopted has aimed at complete exenteration wherever practicable, *viz.*, laryngo-pharyngectomy, partial esophagectomy, thyroidectomy and bilateral neck dissection, followed by either primary, or by secondary, reconstruction. On exploration infiltration was found to be more extensive than preoperative clinical and radiological studies suggested (see Table XIV), so the staging adopted in Table XIII is based on the clinical extent of the neoplasm for non-operated cases, and on the operative findings in those submitted to surgery because exploration is needed to determine the extent of malignant invasion with accuracy.

Unfortunately, even when regional excision has been seemingly complete, reconstruction offers many problems irrespective of the method of repair adopted. Early local recurrence, sloughing of Wookey[14] flaps, postoperative fistula formation

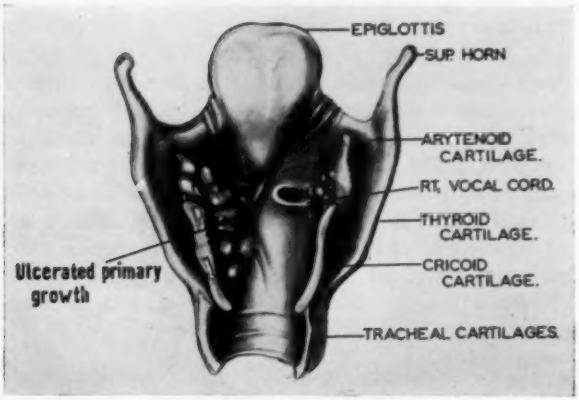

Fig. 5. Glottic variety of cancer of the larynx. This drawing of a laryngectomy specimen shows a tumor arising from the posterior end of the left cord and extending across the posterior commissure to involve the posterior portion of the opposite cord. This type of lesion is classified as glottic.

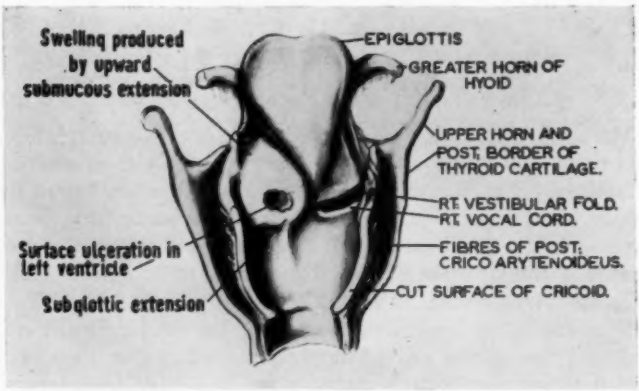

Fig. 6. Glottic type of cancer of the larynx. A drawing of a laryngectomy specimen to show a tumor arising primarily from the left cord. There is submucosal etension to the supraglottic and subglottic regions.

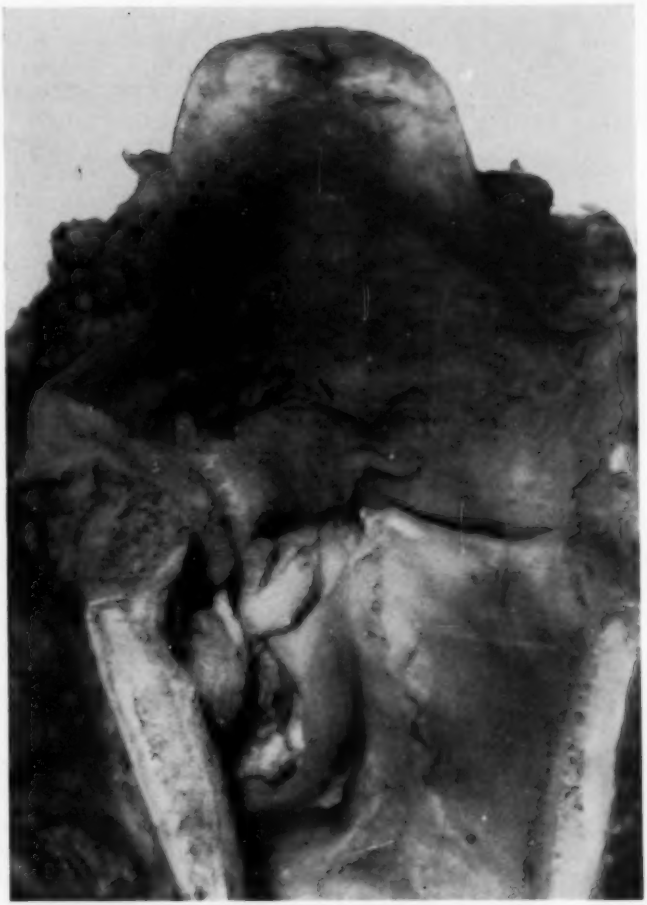

Fig. 7. Subglottic type of laryngeal cancer. This photograph of a laryngectomy specimen shows a typical subglottic tumor arising from the left subglottic space.

and stenosis impose a heavy burden on surgeon and patient alike.

Of 16 laryngo-pharyngectomies six were frankly palliative, in that it was impossible to resect the growth with a sufficiently wide margin of normal tissue due to widespread extension to neck tissues, to the esophagus or to inoperable glands. Even in the remaining ten, the margin of safety was

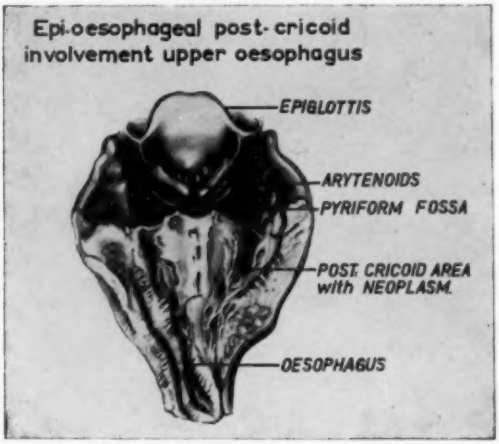

Fig. 8. Epi-esophageal type of cancer of the laryngo-pharynx. This drawing of an operative specimen shows involvement of the hypopharynx and of the cervical esophagus. The exact site of origin cannot be stated with certainty, hence the grouping together of the hypopharyngeal and cervical esophageal varieties under the all-embracing term, epiesophageal.

often far from adequate. In over half of those treated by radiotherapy, treatment was incomplete, too. As luck would have it, the only two patients who were free of disease died of unrelated causes; one by drowning and the other from bronchopneumonia. Careful postmortem examinations were carried out on both. Autopsy proved that there was no malignancy at the time of death.

These gloomy findings do not bear out the more optimistic figures of other authors. There is no selection of cases in the above analysis. Except in hopeless cases, all possibles have

Fig. 9. Glosso-epiglottic carcinoma. This type is not classified with laryngo-pharyngeal neoplasms. A photograph of a specimen showing extensive neoplasm involving the posterior part of the tongue, the valleculae, the epilarynx and the anterior part of the larynx. The epiglottis has been completely destroyed by the ulcerative type of lesion. The epilarynx and larynx are involved secondarily by direct extension.

been given their chance of cure, even when the disease is far advanced. The psychological upset produced by laryngectomy is much greater in the case of the female than in that of the male. In general, women make a poor attempt to adapt themselves, and rehabilitation is a very great problem. The pitiful hurt look of astonished hopelessness on these women's faces after unsuccessful surgery makes one wonder whether the mutilation is worthwhile. Certainly, many bitterly resent

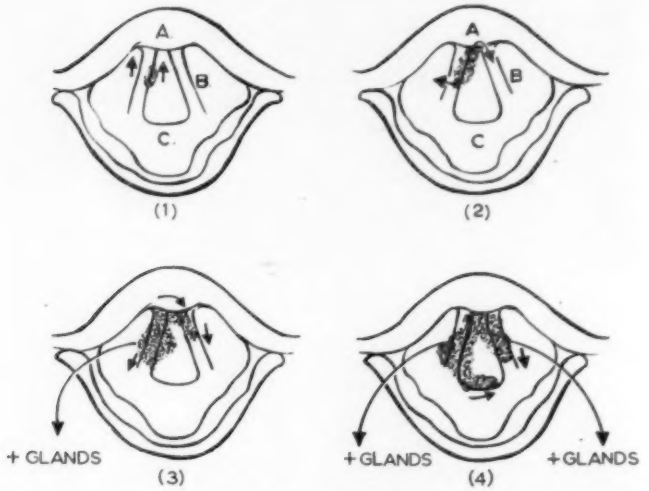

Fig. 10. Clinical classification of cancer of the larynx. The four clinical stages are diagrammatically depicted. Involvement of regional glands is indicated by the letter G. See text for details.

the surgeon's intervention, which can only be justified if a worthwhile prolongation of useful life is achieved; however good the immediate results of a brilliant and faultless technique may be, it is the ultimate outcome that matters, and the conscientious surgeon remembers that his patient's general well-being takes precedence over his surgical ability. If he cannot cure, it should be his aim to reduce and not to increase the sum total of his patient's misery.

One is forced to conclude from personal experience that

regional surgery does not provide the hoped-for answer to the problem of epiesophageal carcinoma unless the cases can be seen, diagnosed, and treated at a much earlier stage than at present, and that radiotherapy has at least as much to offer in the way of palliation. Excluding those dying within the first two months, the duration of life from the beginning of treatment averages six-and-a-half months for patients treated by primary excision and eight months for those palliated by radiotherapy. The patient's well-being is often best served by maintaining nutrition as long as possible, and this may necessitate the insertion of indwelling feeding tubes, or gastrostomy.

PERORATION.

Modern laryngeal surgery owes much to the observations, perseverance and skill of the early pioneers. In this connection I would like to pay tribute to my old chief, the late Dr. Lionel Colledge, who first stimulated my interest in laryngeal carcinoma. Two decades have passed since that time, some experience has been gained but there is still much to learn.

Your Vice-president invited me to deliver this paper, but the honor, and it is a great honor, belongs to my teachers and to my many colleagues. The surgeon is only one member of the team engaged in the battle against carcinoma. On behalf of the others, as well as on my own, I thank you for the honor accorded us.

ACKNOWLEDGEMENTS.

I acknowledge with gratitude the help and cooperation of the Royal Marsden Hospital Staff, particularly Dr. M. Lederman, who has been responsible for the radiotherapeutic treatment of many of the patients; and of my many other colleagues and of those who have kindly referred their cases to the Malignant Clinic of the Metropolitan Ear, Nose and Throat Hospital and to Hillingdon Hospital. My thanks are also due to Mr. E. Stride who has been responsible for the photography, and to Miss B. M. Becket for the drawings for the illustrations.

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PATHOHISTOLOGY OF FETAL EARS AFTER MATERNAL RUBELLA.*†

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Deafness is a well recognized damage to the offspring when the mother acquires rubella in the first trimester of pregnancy. Statistical data are widely divergent, but a general picture of hearing loss is a proven fact.

Numerous clinical observations place the hearing loss into the framework of a rubella embryopathy. Of outstanding importance is the history as to maternal disease and, even more, damage done to other parts of the fetal organism.

Although there is an abundance of clinical experience, pathological data regarding the hearing organ remain scarce. Since the publication of Schall, Lurie and Kelemen in 1951 from this department, Toendury, in 1952, reported on a fetus of 80 days and a premature; Nager, 1952, added three histologically examined cases, and Lindsay, et al., in 1953, five more. It seems that the cases investigated so far are divided equally between embryonic hearing organs and those autopsied in infancy (ten each). The last five years have not brought any new contribution to the histopathology of the hearing organ in rubella.

The four cases presented here came from therapeutical interruptions of pregnancies of mothers who had rubella in early pregnancy. The collaboration of Dr. Kurt Benirschke, pathologist of the Boston Lying-In Hospital, made this material available. His discussions of the antecedents and of the connection with the data of the pathological examination

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†Aided by United States Public Health Service Research Grant B-1272 (C).
Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Dec. 23, 1958.

made it possible to put the ear findings into the framework of general rubellar embryopathy.

CASE HISTORIES.

Case 1—Unit No. 36115. The 23-year-old mother had German measles in the tenth week of her second pregnancy. Hysterotomy was performed and a female fetus of a crown-rump length of 149 mm., corresponding to 17½ weeks of gestation was removed.

No gross malformations were found; the histopathological examination of the brain by Dr. Samuel P. Hicks, resulted in normal findings.

The right temporal bone was sectioned after embedding in paraffin; the left was processed by the usual celloidin technique.

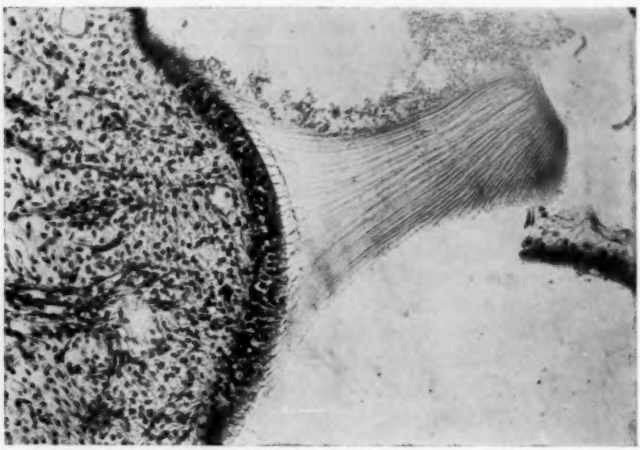

Fig. 1, Case 1. Left side; Heidenhain-Mallory, X250. Ampullar crista, the cupula eroded, one side, by extravasate.

Right Temporal Bone. The tympanic membrane was formed, with the regular strip along its tympanic surface free of mesenchyme, while mesenchyme completely filled the middle ear and the perilymphatic spaces of the inner ear. The ossicles were cartilaginous, with beginning ossification in the long process of the malleus. The mesenchyme of the middle ear showed a considerably looser structure than that of the inner ear. The annular ligament of the stapes was clearly discernible at its medial circumference; laterally the plate was still fastened to its frame, cartilage to cartilage. The periosteal layer of the cochlear capsule was represented by a thin shell, but with intensive osteogenesis along its external surface. The marrow spaces of the endochondral layer were very wide. The finely drawn endosteal layer showed hardly any ossification. The modiolus was still entirely of connective tissue; the endolymph spaces of the vestibulum and cochlea were practically free of mesenchyme.

Maculae and cristae were fully developed, and the organ of Corti, under a rolled-up tectorial membrane, was already a mound elevated above the basilar membrane. The degree of fixation was not sufficient clearly to distinguish cytological details. The membrane of Reissner was in a normal position. The dura was thin and without hyperemia.

Left Temporal Bone. With findings similar to the right side the following can be added. Under the dura, as in a few other locations, a minimum amount of blood was scattered. These small extravasates were, in the inner ear, more conspicuous by their location than by their extent. Even a few red blood corpuscles, when in contact with a cupula, caused destruction of the same (see Fig. 1).

With the ductus reuniens somewhat dilated, dilatation became more conspicuous in the perilymphatic cisterna; sacculus and utriculus were

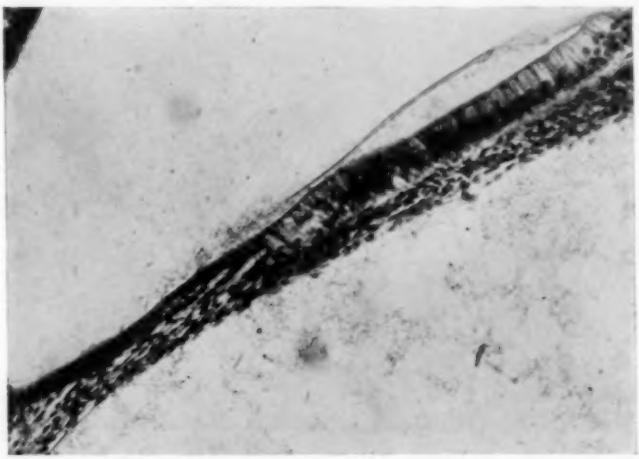

Fig. 2, Case 1. Left side; Heidenhain-Mallory, X250. Organ of Corti in apical turn: row of undifferentiated cells, tectorial membrane adherent.

somewhat compressed by this perilymphatic bulging. The saccus endolymphaticus seemed to be dilated, but, as it was open in the specimen, no deduction could be made regarding its original state. For the same reason, the cochlear aqueduct could not be traced in its entire length. Corti's organ, on both sides lagged in evolutionary degree behind the already fully evolved vestibular end organs. It formed a distinct mound in the basal and in the middle turn, but in the apical turn it still remained an undifferentiated pseudostratified layer of neuroepithelial cells (see Fig. 2). The spiral nerve was already connecting Corti's organ to the spiral ganglion in the connective-tissue modiolus.

Case 2—Unit No. 37932. The 29-year-old mother suffered German measles in the sixth week of pregnancy which was her third. Therapeutic abortion was done at the sixty-ninth day by ovum forceps and curette, with the female fetus removed in pieces. The estimated crown-rump

length was 70 mm., corresponding to nine weeks gestational age. Sections of the placenta and other tissues of the embryo showed degeneration despite persistence of trophoblastic mitoses. The decidua showed areas of necrosis.

In one fragment of the cranial base, parts of the cochlea and the intact inner acoustic meatus were found. The cochlear capsule was entirely cartilaginous without a vestige of ossification. The vestibular and tympanic scalae were filled with mesenchyme, and the cochlear duct was free. The tectorial membrane was smoothly stretched over the organ of Corti. The latter was represented in the apical turn by a straight layer of neuroepithelial cells. In the basal and the middle turns the basal portions of the neuroepithelial cells seemed to be embedded into the substance of the basilar membrane (see Fig. 3), the latter not quite

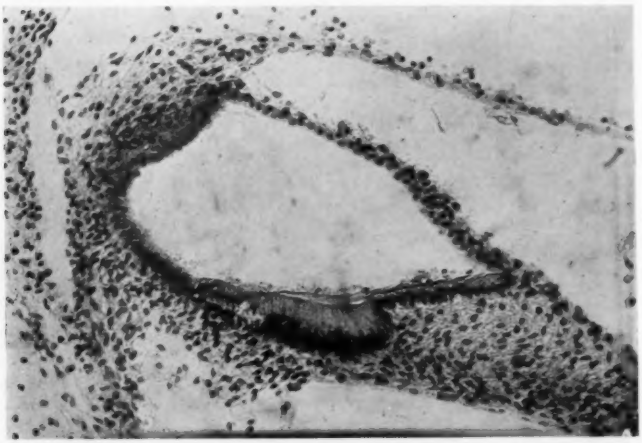

Fig. 3, Case 2. Heidenhain-Mallory, X250. Contents of the cochlear duct, middle turn, free of mesenchyme; mesenchymal masses still in tympanic and vestibular scala.

differentiated from the mesenchymal mass of the tympanic scala. Reissner's membrane was in a normal position. An atypical growth, an epithelial fungus-like formation, was seen in what could be identified as a portion of the utricle.

Case 3—Unit No. 38345. A healthy 25-year-old primigravida had rubella at two-and-a-half months of her pregnancy while working as a domestic in a household in which three children had rubella around this period. The classical eruption was accompanied by retroauricular node enlargement. Abdominal hysterotomy was done in the eighteenth week of gestation. A male fetus of 200 mm. crown-rump length was removed. At necropsy the tissues were macro- and microscopically unremarkable, save for obvious immaturity of a degree consistent with the probable age of gestation. The disparity between measurements and the probably stated date of the conception suggested that miscalculation of approximately one month was made by the mother.

The ears went through the usual celloidin embedding; the right side was sectioned in a vertical, and the left in a horizontal plane.

Right Temporal Bone. The specimen contained only the inner ear and, up to section No. 600, a narrow strip of middle ear. For the remaining 200 sections only the inner ear was present. The ossicles were seen only in part.

Left Temporal Bone. Practically only the inner ear was present.

Both ears showed similar findings which can be described together.

The dura was normal without hyperemia. Very small extravasates were scattered in a few regions but not around the sensory end organs,

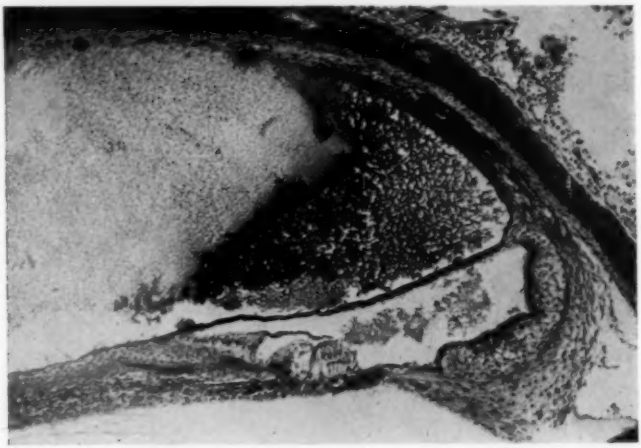

Fig. 4, Case 3. Left side; Heidenhain-Mallory, X125. Fibrinous thrombus in scala vestibuli of middle turn; in cochlear duct still mesenchyme.

and not in the inner meatus. An exception was a small fibrous organized thrombus, not of recent formation, originating from a large vessel in the vestibular portion of the spiral ligament in the middle turn of the left cochlea (see Fig. 4). The subarcuate fossa, filled by mesenchyme, was bridged over by normal dura, which did not descend into the depth of the fossa. The labyrinthine capsule was a solid cartilaginous block although invaded by osteogenic buds. In the capsule of the vestibulum and of the cochlea, ossification was in progression. The periosteal and endosteal layers were formed everywhere. The endochondral layer was a mesh of large vascular connective tissue channels with trabeculae of bone and cartilage and massive invasion of marrow tissue. Osteoblastic activity was intensive in all three layers. Vestibular and cochlear portion of the otic capsule were in the same developmental stage, while the modiolus of the cochlea was still completely formed by connective tissue. Maculae and cristae were fully developed (see Fig. 5). The macula sacculi of the right side was covered by a foamy precipitate that was not present over the three remaining maculae.

The lumen of the saccule and utricule was normal, or somewhat depressed. The evolutional sequence of the organ of Corti was normal. In the basal turn a high mound was formed around the tunnel; in the middle turn (see Fig. 6) the mound was of similar elevation but with less structural differentiation. In the apical turn the arching of the organ of Corti was on its way, with the neuroepithelium still undifferentiated. The tectorial membrane was detached in the basal turn, adherent in the middle and apical turn; at one point (see Fig. 6) it was pulled in the direction of Reissner's membrane by a shrinking mesenchymal band. The mesenchyme disappeared from practically all the endolymphatic spaces. In the remaining regions it showed different aspects. A fibrous

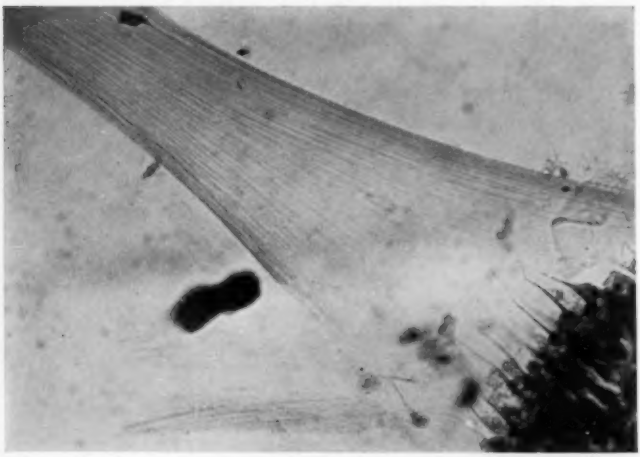

Fig. 5, Case 3. Left side; Heidenhain-Mallory, X450. Crista with cupula, intact.

structure filled the niches of the oval and the round window, while the content of the cisterna under the stapedial plate was an amorphous mass. The scala vestibuli and tympani were filled by the same amorphous type, while both kinds, fibrous and amorphous, were present in the perilymphatic spaces of the semicircular canals. Here the fibrous mesh lined the canal walls, and in the amorphous mesenchymal masses cysts or vacuoles appeared. Conspicuous was the scarcity of cells and vessels. The mesenchyme in the perilymph of the vestibulum suggested the definite trabeculi, and the cysts and vacuoles reminded one of the process of disappearance of the mesenchyme around term.

Ossification started in the ossicles, cortical in the incus, and being present, to some measure, in the stapedial plate. The annular ligament was already outlined, although the plate was still inserted in its frame cartilage to cartilage.

Case 4—Unit No. 38757. This was the third pregnancy of a 28-year-old mother who contracted rubella at about the fourteenth gestational week, with a rash over face and body for two days and an enlarged node in

the retroauricular region. She had no fever and no respiratory or other symptoms. By abdominal hysterotomy a male fetus of crown-rump length of 110 mm. corresponding to fifteen weeks gestational age was removed. On dissection, no gross anomalies were found. The immature placenta showed whole areas of degeneration and, under the microscope, old marginal infarction. The brain was normal. Microscopic examination of the eyes (Dr. Taylor R. Smith) showed rubellar cataracts.

The two ears were obtained in one block, which was sectioned in a frontal plane with resulting paramodiolar sections through the cochlea.

The findings were identical for both sides. Within the middle and the inner ear there was no vestige of ossification, although in the temporal squama it was well progressed.

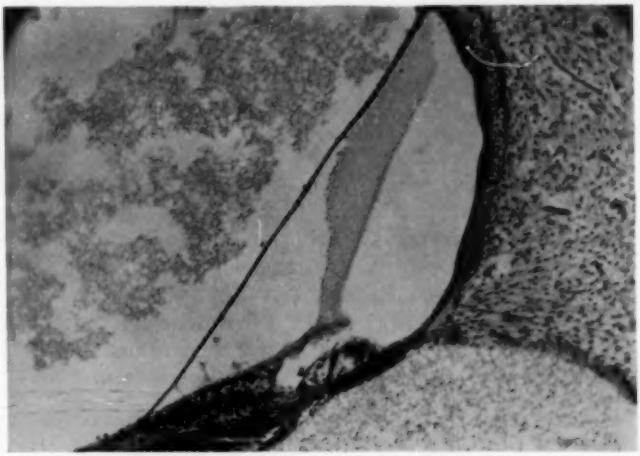

Fig. 6, Case 3. Right side; Heidenhain-Mallory, X175. Organ of Corti in middle turn. Tectorial membrane elevated, like being pulled up by shrinking mesenchymal band; mesenchyme still fills the vestibular and the tympanic scalae.

Ossicles and otic capsule were uniformly cartilaginous, with a connective tissue modiolus in the cochlea. Maculae and cristae were well developed, the latter together with the cupulae. The organ of Corti was represented in the apical turn by a straight row of neuroepithelial cells, while arching of the papilla started in the middle and the basal turn. The nerve connection from the organ of Corti through the spiral ganglion to the VIIIth nerve was formed. The stria was already vascularized to a certain extent. The mesenchyme showed different character in different locations. In the middle ear, including the windows, it was a dense net of bipolar spindle cells, with few vessels; the cisterna perilymphatica was filled with an amorphous, dense, foamy mass. The perilymph spaces of the vestibule contained both the astrocytic and the amorphous type; there were vacuoles in the astrocytic field, which were filled with a mass of the amorphous type. The latter type formed the content of the vestibu-

lar and tympanic scalae, sometimes dense, sometimes rarefied, but always without cells or vessels. The endolymph spaces were practically free of mesenchyme within the entire otic capsule. The tectorial membrane was rolled up over the arching papilla in the middle and in the basal turn.

DISCUSSION.

Technical Remarks. As all material was obtained from surgical interventions, with immediate fixation of the specimen, no postmortem changes could develop. Deficiencies in the histological pictures must all have originated during fixation or other phases of the preparation for microscopy. The staining was done with hematoxylin and eosin, with Heidenhain-Mallory, Gomori impregnation; for polarization unstained sections were mounted. Examination was done in full and polarized light and under phase.

An important point in judging the resulting pictures was the presence of hemorrhage. This was reduced to a minimum, thanks to the careful operative procedures which at the same time excluded any traumatic irritation. Even in Case 2, where the removal was done by curettage, signs of bleeding were absent from the fragment which came to examination. A factor to be remembered in judging the histological picture is the possible effect of a number of drugs administered before the operation and in connection with the anesthesia.

Where temporal bones of children with deafness were examined the functional tests of hearing could give a clue if changes by maternal rubella were present. As this is not the case with fetal material, one cannot be sure whether a case belongs to the group where deafness possibly could have developed. Under these circumstances possible malformations in other organs are of importance. In Cases 1, 3 and 4, the brain showed histopathologically no abnormalities; in Case 4 rubellar cataracts were diagnosed; while the curette case (No. 2) did not lend itself to further observations.

An advantage of the material presented here is its uniformity. The mothers belonged to the same age group, between 23 and 29 years, and rubella had developed at approximately the same gestational periods. Removal was done between the tenth and the eighteenth gestational week, with three cases

in the seventeenth, seventeen-and-a-half and eighteenth week respectively. In three cases both temporal bones were obtained for further study, and in only one was the material reduced to a fragment. The main data are compiled in Table I.

The findings can be discussed around three points of importance: mesenchyme, ossification, and sensory end organs.

The *mesenchyme* showed the same picture throughout the series. In the middle ear there was an astrocytic fibrous

TABLE I.

HOSPITAL NUMBER	MOTHER			RUBELLA	INTERRUPTION		FETUS		TEMPORAL BONE	ANOMALIES OUTSIDE HEARING ORGAN
	AGE	GRAVIDITY	PARA	GESTATIONAL WEEK	GESTATIONAL WEEK	OPERATION	SEX	CR mm		
36115	23	2	1	10	17 1/2	HYSTEROTOMY	♀	148	RT. & LT.	NONE (INCLUSIVE BRAIN)
37932	29	3	2	6	10	CURETTE	♀	70	FRAGMENT	NO INFORMATION
38345	25	1	0	10	18	HYSTEROTOMY	♂	200	RT. & LT.	NONE (INCLUSIVE BRAIN)
38757	28	3	2	14	17	HYSTEROTOMY	♂	110	RT. & LT.	RUBELLAR CATARACT BRAIN NORMAL

mesh, comparatively loose. In the inner ear two kinds of mesenchyme were found: a fibrous mesh enclosing vacuoles filled by an amorphous type in the perilymphatic spaces, with exception of the cisterna under the stapes plate, which was filled exclusively by amorphous, homogeneous masses. The endolymphatic spaces in the vestibule and in the cochlea were practically free of mesenchyme, or showed only insignificant isolated remains of the latter. It is felt that the behavior of the mesenchyme was entirely normal.

The *ossification process* proceeded at the expected rate in all three layers of the otic capsule. The progress corresponded to the gestational period. Case 4 was an exception; here, although the seventeenth calculated week was reached, no

signs of ossification were seen in the cartilaginous walls of the middle and the internal ear, while ossification was already progressing in the squama.

The *sensory end-organs*, together with their neural apparatus, in all four cases were in a stage wholly corresponding to their gestational age. The cristae, including their cupulae and the maculae including the otoconial layer, were already fully developed. The organ of Corti was an undifferentiated neuroepithelial layer in the apical turn, but as it progressed toward the base a more and more conspicuous arching pointed in the direction of development into the definite full mound. The stria showed no signs which could be interpreted as an arrest or even degeneration. The tectorial membrane was still firmly adherent to the surface of the papilla, except where it was rolled back as a result of preparation. With the end-organs of the vestibulum fully formed, and with the organ of Corti showing the expected state of evolution in the different turns, no signs of arrest or degeneration could be noted. Imperfections in the presentation of the Corti papilla could all be charged to the preparatory process, but they could be judged with sufficient certainty to leave no doubt about the evolutional stage.

Hemorrhage might give a clue for possible functional damage within the hearing organ. It can be supposed that the viral attack results in a vulnerability of the vessels resulting in extravasation. One such condition was found in Case 3 in the form of an organized thrombus. Very small extravasates can be deleterious when in strategic locations. The cupulae can be eroded by contact with the smallest amount of blood (see Fig. 1); in the cochlea a minimum extravasation in the basilar membrane underlying Corti's organ, or in the vascular stria, may have far reaching consequences.

Even without exaggeration of the role of extravasates, including their probable disappearance by absorption, it remains to be assumed that a supposed fragility of the vessels will show deleterious consequences at any period of strain, during gestation, around the delivery or in postnatal life. An epithelial formation in the inner ear, as found in Case 2, does not

belong to the rarities and should not be given further consideration.

All in all, insofar as histopathological changes were found within the ear, they are not sufficient to be a cause of functional loss in the peripheral organ. This means that, as in other viral conditions, central neural damage may be present in rubella also.

Toendury emphasized an evident organ-affinity of the rubella virus with destructive effects, the lesions localized in certain structures of the cochlear duct, probably beginning in the stria vascularis. Nager joined in this explanation; a remarkable fact in his tabulation is the presence of otitis media in all infantile cases. Lindsay, et al., found in one of their five cases, a bilateral sacculo-cochlear type of pathology, with thickening and distortion of the wall in the saccule, and adhesions to the macula, with corresponding areas of macular degeneration; and, in the cochlear duct, malformations of the tectorial membrane and Corti's organ, the reduction in the extent of the stria vascularis has been interpreted as an indication of the virus effect, permitting a greater concentration of the virus irritant in the endolymph within the cochlear duct and the saccule.

Schall, Lurie and Kelemen presented as an explanation of arrest of development and vascular lesions a "monistic theory." According to this the vessels are the first to suffer, and the lack of nutrition is followed by developmental arrest; vulnerability of the vessels shows its consequences during pregnancy or any period of strain, at delivery or at any perinatal or postnatal point.

Through careful surgery (hysterotomy) in three of the four cases presented here, hemorrhage was reduced to a possible minimum, with only one region of one case (see Fig 4) showing a somewhat conspicuous extravasate.

Toendury stressed repeatedly, as did others, the difficulty in evaluating normalcy or pathology in the histological picture of the elements of the inner ear. Unquestionably great caution is here in order.

Most of the hitherto communicated cases remained within the frame of normalcy. Assuming the basic importance of vascular damage, the role of the central nervous system is of significance as responsible for the functional loss; however, in three of the present cases the brain was examined and was found to be normal.

SUMMARY.

Four cases of interrupted pregnancy between the tenth and the eighteenth week are reported. The indication for surgery, hysterotomy in three cases and curettage in one, was rubella of the mother in early pregnancy. Both temporal bones were examined in sectional series in the cases with hysterotomy and so was a fragment from the curette case.

Sporadic pathology was found: a massive hemorrhage in a vestibular scala (see Fig. 4), some blood mixed in the mesenchyme of the perilymph (see Fig. 1), band fixation of a tectorial membrane to the membrane of Reissner (see Fig 6).

Adding the present report to a previous one by Schall, Lurie and Kelemen, from this department, six cases failed to show any aural histopathology definitely responsible for the well established fact that hearing loss in the progeny frequently follows maternal rubella.

For the time being the most promising clue may be vascular fragility, leading eventually to abnormalities. In the central as in the peripheral hearing organ, the vascular damage may become a factor at periods of strain: during gestation, at delivery, at any point of perinatal or postnatal life. Study of viral effects on blood vessels may give a clue to a pathological basis for deafness as part of rubellar embryopathy.

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UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The Department of Otolaryngology, University of Illinois College of Medicine, announces two special postgraduate courses to be offered in the Fall of 1959.

ANNUAL OTOLARYNGOLOGIC ASSEMBLY.

The Assembly will be conducted September 18-26, 1959, and will consist of a series of lectures and panels concerning advancements in otolaryngology. Some of the sessions will be devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat. Guest lecturers will participate in an entire day's program reviewing the latest advances and principles of temporal bone surgery.

Chairmen of the Assembly are Maurice F. Snitman, M.D., and Emanuel M. Skolnik, M.D.

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

The course in laryngology and bronchoesophagology, under the chairmanship of Paul H. Holinger, M.D., is scheduled November 9-21, 1959.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

CONSIDERATION OF CHANGE IN PRESENT POLICY OF PERFORMING TONSILLECTOMIES AND ADENOIDECTOMIES DURING SUMMER MONTHS.*†

ALBERT B. SABIN, M.D.,
(By Invitation),

Cincinnati, O.

The question before us is whether the widespread use of Salk vaccine and the resulting diminution in the incidence of paralytic poliomyelitis justify a change in the present policy of prohibiting the performance of elective tonsillectomies and adenoidectomies during the Summer months, or so-called "poliomyelitis season." First, we shall consider the grounds on which these operations have been prohibited and then analyze the manner in which the factors under consideration may have been altered by the use of Salk vaccine.

These operations have been prohibited during the "poliomyelitis season" because of an increased risk of development of bulbar poliomyelitis. This increased risk is believed to be due to:

1. Increased dissemination of polioviruses during the "poliomyelitis season."

2. Actual contamination of severed pharyngeal nerves with virus either already present in the throat at the time of the operation, or localizing in the wound from the blood shortly after the operation.

The first question that we must ask, therefore, is whether there is any evidence that the widespread use of Salk vaccine has diminished the dissemination of polioviruses during the "poliomyelitis season." The answer is that all the data gathered thus far indicate that the antibody produced by Salk vaccine does not prevent extensive multiplication of the virus

*Presented at Meeting of Eastern Section of American Laryngological, Rhinological and Otological Society, Inc., New York, N. Y., Jan. 8, 1959.

†Personal studies reported herein were aided by grants from The National Foundation.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 19, 1959.

in the intestinal tract, and that dissemination of the virus is readily accomplished by children with an adequate immune response to the vaccine. Earlier experimental[1,2] and field studies[3,4,5] were carried out in persons who had received only one or two doses of Salk vaccine. During the past year we[6] have completed a study of 26 children, whose antibody status was known prior to the inoculation of four doses of Salk vaccine over a period of 14 months, and who were subsequently fed attenuated polioviruses. This study showed that the immunity produced by the four doses of Salk vaccine had no effect either on the amount or duration of virus excretion in the stools, and that these immune children were capable of infecting other fully vaccinated children who were in contact with them in an institution under conditions particularly suitable for fecal contamination. Recent studies by Fox, Gelfand and their associates[7] established the capacity of children, who had received three doses of Salk vaccine, to disseminate naturally acquired polioviruses as well as orally administered attenuated strains under ordinary home conditions.

Regarding the special problem of tonsillectomies and adenoidectomies, we are especially interested in how polioviruses localize and multiply in the throat, and how the immunity produced by Salk vaccine may influence either the localization or the multiplication of virus in the posterior pharyngeal wall. Studies carried out in 1955 indicated that poliovirus could be demonstrated in the posterior pharyngeal wall of 83 per cent of a group of 23 patients with clinically diagnosed poliomyelitis tested within two to six days after onset of first symptoms.[8] This study also established the absence of virus multiplication in the mouth and nose where only traces of virus were found in four of this group of 23 patients. Experimental studies on human volunteers who swallowed various strains of attenuated polioviruses, established the fact that with viruses, which have no demonstrable viremic phase, the localization and multiplication of virus in the throat is a function of the amount that is swallowed.[1c,8a] The word swallowed must be emphasized here, because different results can be obtained when the virus is swabbed on the throat or squirted directly on it. To obtain an incidence of about 80 per cent localization

in the throat, such as was found in the patients with clinical disease, it was necessary to feed one million or more tissue culture infective doses of the attenuated strains. The question, therefore, arises whether the patients with naturally acquired clinical disease actually swallowed such large amounts of virus, or whether the high incidence of virus in their throats may be accounted for by another mechanism. The maximal amounts of virus excreted by naturally infected patients, as well as by children experimentally infected with attenuated strains, is about one million tissue culture infective doses per gram of stool.[1c,8a] A person would, therefore, have to swallow at least a gram of stool to obtain such a large dose, which seems most unlikely. The amount of virus that can be obtained by swabbing the throat at the peak of multiplication is in the range of 1,000 to 100,000 tissue culture infective doses, and the absence of virus from the mouth, saliva and nose except in trace amounts in rare instances, indicates that the virus in the throat cannot be a source of infection under normal circumstances. While it is possible that with virulent epidemic strains, less virus may be necessary to initiate multiplication in the throat, it is also possible for such strains to localize in the throat secondarily after they have multiplied in the intestinal tract and other extraneural tissues and produced a viremia. For viruses localizing in the throat secondarily from the blood stream, even minimal amounts of antibody produced by Salk vaccine should be sufficient to block such localization.

In a study carried out by Wehrle, et al.[9] on familial associates of paralytic cases, virus was recovered from 6 per cent of 52 pharyngeal cultures obtained from 14 children who had received one or two doses of Salk vaccine, as compared to 17 per cent of 88 pharyngeal cultures from 23 unvaccinated children.

Davis et al.,[4] who carried out a similar study during the 1956 Chicago epidemic, limited the analysis of their results to ten families in which at least one member was found to have virus in the throat. Among 16 unvaccinated children, virus was recovered from the throat of eight, while among 16 vaccinated children only four yielded virus—one virus

isolation was obtained from six children who had one dose, three isolations from seven who had two doses, and none from the three children who had had three doses. While the interpretation of such studies is limited by the fact that one does not know the immune status of the unvaccinated control group or of the vaccinated children prior to inoculation of Salk vaccine, they, nevertheless, show that under natural modes of transmission, virus can be found in the throat of some children who have had only one or two doses of Salk vaccine.

Horstmann et al.,[2] who fed 25 million tissue culture infective doses of the Type 1, L Sc. attenuated strain to children immunized by two doses of Salk vaccine, demonstrated viral multiplication in the throat of six of the seven children tested. It should be noted, however, that the dose of virus was very large, and with one exception the titers of antibody were 16 or less. It is known that for antibody to appear in the nasopharyngeal secretions the titer in the blood needs to be in the range of 32 or higher. During the course of our study[6] on children who had received four doses of Salk vaccine, we tested for virus in the throats of nine children, four of whom received about six million tissue culture infective doses of Type 1 attenuated virus, and five about 2.5 million doses of Type 3 in a teaspoonful of syrup (see Table I). On the basis of other tests in adult volunteers, without homotypic antibody, one could have expected to find virus in the throat of about 80 per cent after ingestion of such a dose; however, despite repeated tests no virus was recovered from the throat of any of these children, although all of them excreted virus in the stools for a period of two weeks or longer. Since two of these children had no demonstrable antibody and one had only a titer of four at the time of virus feeding, and yet also had no demonstrable virus in the throat, one may question the significance of the negative findings in the other six children with high titers of antibody. Fox, Gelfand and their associates[7] used aliquots of the same attenuated strains in a study on 16 unvaccinated medical students without homotypic antibody as well as on 14 who had received three doses of Salk vaccine. Although their study is not comparable to ours, because they squirted the virus directly on the throat, the results, nevertheless, indicated that pharyngeal infection oc-

curred only in those whose antibody was either not demonstrable or in titiers not higher than 1:8 by their method of testing. Thus, in their tests with Type 1 the virus multiplied in the throat of all six unvaccinated students without homotypic anitbody, and in only two of six vaccinated students who excreted virus in the stools after receiving 32,000 to 800,000 tissue culture infective doses.

The conclusion appears to be warranted that when Salk vaccine of *sufficient potency and dosage* is given to produce

TABLE I.

Presence of Poliovirus in Throat Swabs and Stools from Children, Who Lacked Indicated Type of Antibody Prior to Receiving 4 Doses of Salk Vaccine During a Period of 14 Months, and Then Were Fed Large Doses of Attenuated Poliovirus 3 Months After the Fourth Dose.

Virus Fed	Name	Antibody Titer just before Feeding	Presence of Virus in		Antibody Titer after Feeding
			Throat Swabs	Stools	
Type 1	Kee.	<4	0*	+	512
(L Sc, 2 ab)	O'Ha.	128	0	+	2048
$10^{6.8}$ TCD_{50}	Bac.	256	0	+	1024
	Seh.	256	0	+	2048
Type 3	Abb.	<4	0	+	1024
(Leon, 12 a,b)	Bar.	4	0	+	512
$10^{6.4}$ TCD_{50}	But.	64	0	+	256
	Cam.	64	0	+	4096
	Gau.	64	0	+	256

0 = No virus detected in swabs taken 3, 5, 7, 10, 14, 21, and 28 days after feeding of virus.
+ = Virus multiplication for 2 weeks or longer.

an adequate antibody response, one may expect to prevent localization of virus in the throat either by prevention of deposition from the blood stream, or by neutralization at the time of primary implantation. Since the increased risk of tonsillectomies and adenoidectomies during the "poliomyelitis season" is believed to result either from the presence of virus in the throat at the time of operation or from subsequent localization in the wound of virus that might be circulating in the blood, one may be justified in carrying out such operations on those who have been successfully immunized with Salk vaccine; however, in drawing up specific recommendations regarding those who may or may not be submitted to

TABLE II.

Antibody Status Among Children, Originally Lacking Indicated Type of Antibody, at Different Times During and After Inoculation of 4 Doses of Commercially Produced Salk Vaccine Between Nov. 7, 1956 and Dec. 23, 1957.

Antibody Type Absent Prior to Salk Vaccine	No. of Children Tested	Per Cent Without Antibody in Indicated Category at Indicated Time.					
		Category	1 month after 2nd Dose	1 month after 3rd Dose	5 months after 3rd Dose	2 weeks after 4th Dose†	3 months after 4th Dose
1	9*	No Antibody	56	22	44	12	25
		<32	78	22	78	25	25
3	19	No Antibody	47	42	53	0	11
		<32	68	63	84	11	44

*One of these children did not receive the 4th dose and the percentage incidence after the 4th dose is based on 8 children.

†An especially selected lot of vaccine of high antigenic potency was used for the fourth dose.

NOTE: The antibody results given here were obtained by the highly sensitive pH (metabolic inhibition) test. The incidence of negative results and low titers was much greater when delayed readings in the cytopathogenic test were used.

such operations during the "poliomyelitis eason," one is faced with the decision as to what constitutes adequate immunization with Salk vaccine. The Salk vaccine produced commercially since the adoption of the more stringent safety tests has often been of poor potency, especially for Types 1 and 3.[10,11] The results that we[6] obtained in a group of five to ten-year-old children, who received four doses of commercially produced Salk vaccine between November, 1956, and the end of December, 1957, prior to a test on the effect of feeding attenuated polioviruses are summarized in Table II. It should be noted that with one exception all of these children already had naturally acquired immunity to one or two types of virus and, therefore, would be expected to respond better to Salk vaccine of poor potency than "triple-negative" children. It can be seen that even three doses failed to produce antibody in a considerable proportion and that five months after the third dose, 44 per cent were without demonstrable antibody for Type 1 and 53 per cent without Type 3. When a serum titer of 32, the minimum level that may be expected to yield detectable antibody in the nasopharyngeal secretions, was taken as the standard, 78 per cent in the Type 1 group and 84 per cent in the Type 3 group had titers of less than 32 five months after the third dose. The fourth dose of vaccine, consisting of an especially selected lot of high antigenic potency, produced an antibody response in all but one of the children within two weeks, which in a few instances disappeared again within three months.

In the light of these considerations and results, I drew up the following recommendations regarding tonsillectomies and adenoidectomies during the "poliomyelitis season," which were adopted by the Cincinnati Board of Health and followed during the 1958 season:

Emergency operations on the throat can be performed during the so-called "polio season" without reference to the vaccination status of the patient.

For *elective* tonsillectomies and adenoidectomies, the term "elective" is understood to mean an operation that can be postponed for a period of at least two weeks without harm to the patient.

In such cases, operations can be performed during the "polio season," providing that the patient has had at least three inoculations of polio vaccine, the last dose having been administered not more than six months prior to the date of operation.

If the interval between the last dose of vaccine, regardless of the number of doses previously given, and the date of operation is greater than six months, another dose of vaccine should be administered two weeks prior to the operation.

It was also agreed that if a high incidence of poliomyelitis appeared in the community, these recommendations may be withdrawn. The main objection to these recommendations may be the unpredictability of the immune status of triply-negative children six months after three doses, or two weeks after a fourth or fifth dose of Salk vaccine of current manufacture. I assume that laryngologists prefer the Summer season for necessary elective tonsillectomies or adenoidectomies not only because of convenience, but also because of the decreased risk of severe postoperative infections that are more likely to occur during the cold months of the year when polioviruses disseminate less but pathogenic bacteria—not all of them readily controlled by antibiotics—more. The physician is thus faced with the problem of weighing risks.

In checking on the number of tonsillectomies and adenoidectomies that were performed in Cincinnati hospitals during the period of June 1 to November 1, I found that 684 such operations were performed in 1957 before the above-mentioned recommendations were adopted, and 1,315 in 1958, when the hospitals required a certificate indicating that the patient had been vaccinated in accord with the recommendations. It seems to me that even though the proposed number and intervals of Salk vaccine injections cannot assure the desired immune status in all children, it was better to have this additional protection than to perform such operations without reference to the vaccination status of the patient as was done in Cincinnati in 1957, and undoubtedly in other parts of the country as well. In view of the 1958 experience in the United States, indicating that a certain proportion of the reported paralytic cases occurred in children who have had three doses

of Salk vaccine, and in view of the fact that no action has as yet been taken to provide Salk vaccine of uniformly adequate potency, I would change my 1958 recommendations to require a minimum of four doses of vaccine, and still another dose two weeks prior to operation if the interval since the last dose is three months or greater. I would still favor prohibition of such operations in communities in which poliomyelitis is epidemic. It should be pointed out that polioviruses disseminate also during the cold months of the year, although at a lower rate than during the Summer months. Accordingly, when operations on the throat are performed during the cold months of the year, the risk of a complicating bulbar poliomyelitis is also not eliminated with complete certainty.

Some may not agree with the requirement of at least four doses of vaccine and the inoculation of another dose two weeks before the operation, if the interval since the last dose is three months or greater, because they believe that the currently available Salk vaccine is usually better than our data indicate, while others may be opposed to any change in the present policy of performing these operations during the Summer season until uniformly effective vaccine is available.

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INSTITUTE OF OCCUPATIONAL HEARING LOSS.

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This course presented by Colby College for seven consecutive years is designed to train otologists and others interested in this problem in initiating and conducting hearing conservation programs in noisy industries. In order to allow for maximum individual instruction, course enrollment is limited. The diversity of experience of the faculty members, assembled especially for this course, provides an unusual opportunity for personal consultation on any aspect of occupational hearing loss.

The dates of the course are August 17-22, 1959, and the fee of $200 includes tuition, board and room. Requests for further information should be made to William A. Macomber, Colby College, Waterville, Maine.

OUTLINE OF THE MANDIBULAR JOINT SYNDROME.*†‡

JAMES B. COSTEN, M.D.,

St. Louis, Mo.

Identification of the mandibular joint syndrome[1] as a symptom complex involved sorting out its qualities from the maze of known reactions of Vth nerve pain.

This search began when enormous interest in otolaryngology was centered about the sphenoidal sinus and its problems. Similarity in distribution of ear and vertex pain directed attention to the temporo-mandibular joint.

The otolaryngologist, once interested, found many such cases because of the location of symptoms. The neurosurgeon welcomed proof of such a secondary neuralgia, to explain the origin of pain reactions which were clearly not tic douloureux.

The sphenoidal sinus has not been declassified as unimportant. Its position of interest was simply scaled down to a secondary place by increased knowledge of allergy, nasal physiology and antibiotics.

Otalgia, vertex pain, glossodynia,[2] and varieties of trismus[3] which activate the mechanism, are clearly established in diagnosis of this symptom complex.

A majority of dental groups showed marked enthusiasm for this syndrome, some parts of which had appeared years ago in the dental literature. It was refused by others because of disagreement with the anatomical reasoning, or because of misunderstanding of the purport and meaning of the study.

There is general acceptance, however, of the origins of painful reflexes as follows: *1.* Abnormal condyle movement pro-

*Read before the Middle Section of the American Laryngological, Rhinological and Otological Society, St. Louis, Mo., Jan. 13, 1959.

†From the Department of Otolaryngology, Washington University School of Medicine, and the Oscar Johnson Institute.

‡This work was supported entirely by the John S. Swift Fund.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 15, 1959.

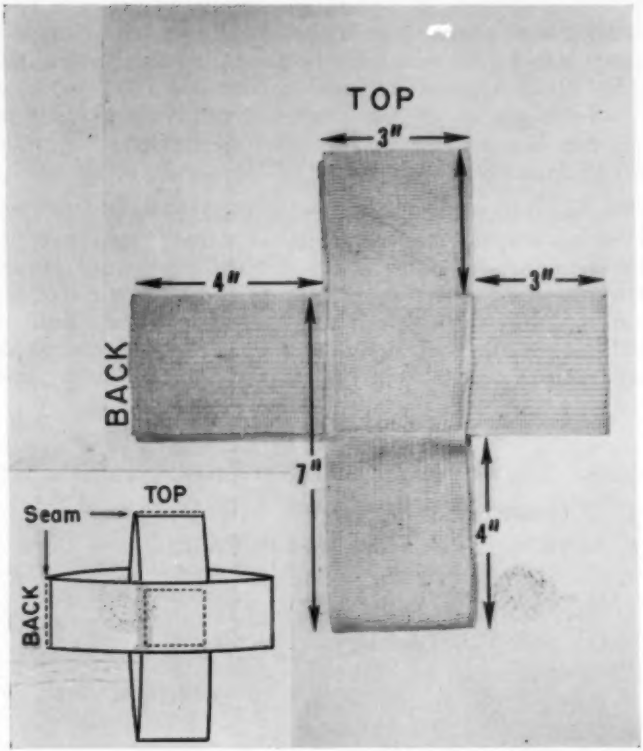

Fig. 1. This type of elastic chin support is the most practical for "splinting" the mandible, in controlling painful movement of the condyle. Change from the condyle's habitual rest position is made by interposing a small cork disc between teeth opposite the painful side, or bilaterally if both sides are affected.

The size illustrated may be used for the average head, adding ½-inch to both vertical and horizontal loops for large, and using ½-inch less for smaller size ranges. The supports are best produced in hospital linen rooms. The material is: 3-inch Natural Color Elastic, Style No. 3746, produced by United Elastic Corp., Stuart Division, Stuart, Virginia.

ducing sensory irritation of branches of the auriculotemporal nerve which distribute to the capsule of the joint; *2.* Direct trauma to the chorda tympani[4] nerve which passes along the medial wall of the glenoid fossa; *3.* and direct trauma to the retrocondylar pad,[5] known to contain sensory elements. In addition to pain in these distributions, reflex pain occurs in

the lingual nerve with typical glossodynia. Once pain is initiated, reflex contraction of the masseter group of muscles perpetuates the trismus cycle by producing further impingement. Much confusion could be avoided in correct evaluation of cases suspected of mandibular joint involvement, if the following simple classification were kept in mind; the groups overlap in many cases.

The commonest source of diagnostic error is a hidden cause of sensory stimulation which is unrecognized; or a conversion neurosis case who clings to the original symptoms, refusing to admit improvement after treatment, dental or direct, has been applied. The common error is to repeat treatment on the basis of positive local findings, when no improvement has been gained.

CLASS I.

Temporary, with otalgia and trismus effects, simple origin:

1. Ulcerated teeth.
2. Excessive yawn and condyle subluxation.
3. Stretch of jaws under anesthesia.
4. Blow on chin.
5. Gripping of jaw during extreme pain.
6. Parotitis, early parotid tumors.
7. Furuncle or cellulitis of external ear canal.
8. Quinzy.
9. Injection of local anesthesia preceding dental treatment.
10. Tetanus.

Treatment of diseases of the temporomandibular joint, embraces everything from removal of a single source of trismus to surgical attack upon the joint itself. If an accidental movement of the lower jaw, such as a blow to the jaw or a yawn, produces enough irritation within the joint, trismus and otalgia ensue. The treatment is external elastic splinting, local heat and sedative drugs. If the origin is an abscessed tooth or infection of the ear canal, removal of these sources is the solution.

Overclosure of jaws with compression of eustachian tubes

proved to be the only factor in deafness. This was a subjective reaction of the patient and not substantiated by audiometric tests. *The responsibility of the otologist is to oppose strongly any advice that promises that change of occlusion will benefit hearing.*

CLASS II.

Semi-permanent, recurrent, with pain effects and structural changes:

1. Fracture of the jaw.

2. Intermittent trismus from third molar impaction.

3. Abnormal joint relations in development of asymmetric jaws.

4. Erosion of condyle, meniscus, and tubercle, as a malocclusion effect.

5. Sharing general osteoporosis.

If the joint changes are secondary to long standing malocclusion and improper action of the condyle, irreversible changes in the meniscus, cartilage and bone structures of the joint occur. These tissues cannot be improved, but restoration of balanced occlusion usually removes the source of stress and damage, so that pain and trismus are relieved.

So many cases of this group improve on splinting of the jaw, concurrent with injection of the joint with hydrocortisone, that restoration of occlusion becomes optional. Interruption of the trismus cycle thus produces such comfort that no other treatment is necessary, unless grossly bad action of the condyle continues the irritative reflex and a pain sequence again ensues.

Examples selected from this class, in which occlusal restoration has failed, or succeeded temporarily, respond to resection of the meniscus and retrocondylar pad. The meniscus is usually found to be deformed, rough with dense adhesions, or impacted upon exotoses of the condylar head. The retrocondylar pad, bearing sensory elements, is resected along with the meniscus. Pain is relieved, but excursion of the condyle is neither restricted nor improved by the procedure.

CLASS III.

Semi-permanent, recurrent, with or without joint pathology:

1. Bonafide pain effects relieved by occlusal restoration, but retained by patient as a conversion neurosis.

2. Hysterical trismus with no joint changes.

3. Pain effects, tiring of jaws, inability to tolerate dentures, based on masseter tremor.

During the study of a group of cases unimproved after dentistry, masseter muscle tremor,[6] an obscure muscle reaction with psychogenic implications, was identified as important in the etiology of these unimproved cases. Ordinarily, tremor has been associated with fear, shock, and neurologic changes. Apart from these conditions, it had usually been unnoticed when the mandibular joint syndrome was studied. The pathogenesis is not clear, neurological examination being negative.

Since emotional overlay is a large factor in this condition, psychotherapy appears to hold promise in treatment. Meanwhile, in spite of the failure of treatments directed to jaw structures, the patients, without exception, are relieved by passive support, simple elastic splinting of the jaw and masseter muscle group. Short half-hour periods are more effective and more restful than longer ones. A few have shown improvement on tranquilizing drugs, small doses being given along with other measures.

CLASS IV.

Permanent, developing ankylosis:

1. Arthritis pain effects, subsiding as ankylosis increases.

2. Fibrosis after fracture of head of condyle or tympanic plate.

3. Fibrosis and osteitis after mastoiditis, a delayed effect of one year or more.

4. Foreign body, as bullet injury.

5. Fibrosis of internal pterygoid or masseter fascia after dental anesthesia (injection), and after parotid abscess.

When excursion is restricted, partially or completely, as in ankylosis, amputation of the condyle is done. All fibrous scarring about the joint and coronoid process is resected. When matted and firm, removal of the coronoid process is sometimes necessary before free movement of the lower jaw is obtained. When a partial or limited condylar resection is anticipated, the trouble-free endaural incision[7] is used to expose the joint. Incision anterior to the auricle, and along the zygoma may be readily used for more extensive exposure of the ramus.

Unnecessary surgery on one or both joints has converted some patients belonging to the neurosis group, Class III, into mutilations of Class IV.

SUMMARY.

It is not necessary to revamp our reasoning as to why the mandibular joint syndrome occurs. The findings are mainly objective and obvious.

It is extremely important to apply direct measures to the joint action, such as elastic splinting of the jaw and injection of steroid substances. These should be used routinely, over long periods, even in the presence of suspected neuroses.

Four general categories have been mentioned. In all groups the complex interaction of neuromuscular cycles requires the use of measures which remove the source of trouble, and still leave jaw function.

Relief of pain is primary in all treatment, but as in no other joint in the body, further ankylosis must not be implemented in the course of treatment.

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OTOLOGICAL FUNCTIONAL MICRO-SURGERY POST GRADUATE COURSE.

A practical course of surgical techniques will be given in English, from July 1-10, 1959, at the University of Bordeaux, France. Dr. Michel Portmann, Associate Professor, will have charge of these courses.

During this period, every assistant attending the course will be enabled to observe surgical interventions in the theater, to follow theoretical courses illustrated with films regarding the major techniques, and to work on his own daily anatomical specimen with all the requisite material, including binocular microscope. The number of assistants is limited to ten. Fees: $100 (to be paid at the Faculty of Medicine in Bordeaux at the beginning of the course).

Applications should be sent to: Dr. Michel Portmann, 45 Cours Foch, Bordeaux, France.

TUMORS OF THE NASOPHARYNX.*

Case Reports.

R. W. HANCKEL, M.D.,

Charleston, S. C.

Tumors of the nasopharynx may be classified as benign or malignant. The benign variety is occasionally malignant in effect by reason of position, as will be illustrated. The most frequent benign lesion is the juvenile nasofibroma; others are neurofibromas, lipomas, chondromas, and teratomas.

Examples of the most frequently found malignant tumors are: *1.* Squamous cell carcinoma; *2.* anaplastic carcinoma; *3.* lympho-epithelioma; *4.* salivary gland tumor; *5.* lymphoma, and *6.* plasmocytoma.

Malignant growths of the nasopharynx are frequently so anaplastic that classification is often a matter of opinion.[1] One radiotherapist[2] classifies all malignant tumors of the nasopharynx as those of limited radiosensitivity and those of marked radiosensitivity. The first of these is squamous cell carcinoma, and the second is the anaplastic variety, which includes all the others.

It is also interesting to note that the nasopharynx, being as inaccessible as it is, and the primary tumor frequently being only a small non-ulcerated elevation of the mucosa, not only the clinician during life, but also the pathologist at autopsy has difficulty in identifying the primary site of the tumor.

It is not surprising then to find that in three series of cases (Gotfredsen,[3] Simmons and Ariel,[4] and McWhirter[5]) the average elapsed time between the first visit to the physician and the discovery of the primary site was ten months. This regrettable lapse of time constitutes the chief reason for writing this paper. We hope that by focussing attention to tumors in

*Read at the Meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., Jan. 23, 1959.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 28, 1959.

this area we will be able to alert at least some members of the medical profession who will be able to make a diagnosis before the condition has progressed to the stage of cervical node metastasis.

The age of the malignant cases varies from 16 to 80 years. One author (Schindler,[6] et al.) reported a teratoma in the nasopharynx of a newborn, which was removed successfully when the patient was only 18 hours old. Juvenile nasofibromas occur usually a few years prior to and during puberty. They may or may not tend to regress in the mid-twenties. The preponderance of malignant cases is in males by a ratio of almost 2 to 1. There is a much higher incidence of malignancy among the Orientals, especially those from the southern provinces of China.[7] This racial predisposition is present among the Chinese, whether they are in their native country or have been transplanted to other lands. The statement has been made that if one passes in the street a cross-eyed Chinese with a swelling in his neck the diagnosis of a carcinoma of the nasopharynx can practically be made in passing.

SYMPTOMS.

Benign tumors present obstructive symptoms locally in the nasopharynx, producing either nasal obstruction or eustachian tube obstruction, or both.

Malignant tumors present a variety of symptoms which may lead the patient to a general practitioner, a neurologist, an ophthalmologist, an otolaryngologist, or a dentist. These symptoms may be conveniently grouped into three categories: *1.* Those attributable to the primary tumor; *2.* those which result from direct extension of the tumor outside the nasopharynx; *3.* those which result after metastasis to the regional lymph nodes occurs.

1. Findings due to the primary tumor[8]:

These consist of a variety of commonplace complaints, and this is one reason that a diagnosis is not made earlier. These are in the order of frequency—nasal obstruction, deafness, unusual post-nasal discharge, which is sometimes blood-tinged, and occasionally tinnitus.

2. Findings due to extension outside the nasopharynx[8]:

The motor nerves of the eyes are most frequently affected. The abducens nerve is the most vulnerable, and diplopia and a convergent squint are frequent findings. The Vth, IIIrd, and IVth cranial nerves, in that order of frequency, are also affected and produce visual disturbances, ocular paralysis, facial pain, headache, and trismus. When the tumor encroaches on the juglar foramen the IXth, Xth, XIth and XIIth cranial nerves, and the cervical sympathetic trunk become involved, producing Horner's syndrome and paralysis of the tongue and vocal cord. With such a diversity of nerves involved it is small wonder that the patient becomes confused as to whom to seek in his dilemma. Even the physician, or dentist whom he seeks, is frequently confused, and many sound teeth are extracted and many unnecessary operations performed in a vain effort to remedy his unresolved complaints.

The tumor may extend up the eustachian tube and produce tinnitus, deafness and serous otitis. In some cases the middle ear has been invaded and material for biopsy has been taken from the external auditory canal.

3. Findings after extension to regional lymph nodes[8]:

The nodes most frequently involved are those high up in the posterior cervical chain. They are usually soft and quite large. The enlargement is usually more advanced on one side than on the other.

DIAGNOSIS.

Examination of the nasopharynx may be accomplished by inspection, palpation, X-ray examination, or any combination of the three.

Inspection may be done by using a nasopharyngoscope, a Yankauer's speculum or a postnasal mirror. By using a soft rubber catheter, the two ends of which are threaded through the nostrils and the loop drawn up against the columella as the ends are drawn through the mouth, the soft palate may be elevated. A postnasal mirror may be used after elevation

of the soft palate by the catheter. Local anesthesia in the form of a spray or nasal packs may be used to facilitate these procedures.

Palpation may be accomplished by inserting a finger into the nasopharynx under either local or general anesthesia. Occasionally it is necessary to split the uvula and soft palate while the patient is under anesthesia to facilitate inspection.

Roentgenograms of the region are often quite helpful. Lateral views and occipto-mental views are particularly useful. The lateral views may be taken with the head erect or with the patient lying on his back and his head over the end of the table. In the latter position, iodized oil is instilled into the nasopharynx and may show a filling defect. The occipto-mental view is of particular value in showing erosion of bone at the base of the skull.

Cytological examination of nasal and choanal washings is a diagnostic aid of some value.

Diagnosis is confirmed by biopsy. Tissue may be difficult to obtain, and occasionally the process is accomplished only by blind biopsy, in which the biting forceps are introduced along the floor of the nose.

PROGNOSIS.

If the tumor is malignant and of the anaplastic, radiosensitive variety, and is of limited scope, the outlook is fairly promising if irradiation is begun promptly. If the tumor is a squamous cell carcinoma of limited radiosensitivity the prognosis is not so good.

If bone erosion has occurred the mortality is almost 100 per cent.

If the tumor is a juvenile nasofibroma which fails to respond to the usual remedies, it may extend intracranially and produce fatal results.

TREATMENT.

Irradiation of the radiosensitive tumors of the nasopharynx is the treatment of choice.

Those tumors of limited radiosensitivity, *i.e.*, the squamous cell carcinomas and the juvenile nasofibromas usually require a combination of X-ray therapy followed by surgical extirpation. The juvenile nasofibromas should also be treated with testosterone.

The strictly benign tumors are removed surgically.

In the author's opinion, the trans-palatine procedure offers the best approach to this area.

CASE REPORTS.

The first three case reports have to do with juvenile nasofibromas and show how greatly they can vary in degree of involvement.

Case 1. The first is that of L.H., whose diagnosis was made in 1953 at the age of nine years. Attention was first attracted to his condition by a recurring nosebleed. During the next five years there were three attempts at surgical removal and four courses of X-ray therapy. At his last visit, in November, 1958, the tumor had almost completely disappeared, and he was playing football and leading a normal life for his age.

This case is presented to illustrate what may be expected of a nasofibroma which responds to the usual therapy.

Case 2. The second case is that of T.K., whose nasofibroma was discovered during the course of a routine adenoidectomy in September, 1943, at nine years of age. It was incompletely removed at this time and recurred. The patient's family became converted to the Christian Science faith and refused to allow further medical or surgical interference. The tumor continued to grow for a number of years, and invaded the nasal cavity and the right antrum. Meanwhile the patient had joined the Armed Forces and was stationed in Tokyo. In October, 1955, at the Tokyo General Hospital he had a radical extirpation of the tumor. There had meanwhile developed a considerable facial deformity. The tumor completely disappeared and the facial deformity is the only permanent defect that he has.

This case shows what comes to pass when a nasofibroma is neglected for a number of years.

Case 3. The third case is that of W.S., whose nasofibroma was first discovered in October, 1957, when he was 14 years old. He was given a course of X-ray therapy, and an attempt at surgical removal was made in January, 1958. This was only partially successful, and the tumor developed rather rapidly. In June, 1958, another attempt at removal was made, including splitting the uvula and soft palate to facilitate a more adequate exposure. The left external carotid had to be tied off at this time to control hemorrhage.

Just before operation a swelling had appeared in the left temporal area. This increased in size after operation. The tumor in the nasopharynx again increased rapidly in size in spite of Roentgen therapy and testosterone. After considerable investigation, including angiograms it was decided to try to remove the tumor surgically, including the extension to the left temporal area. This was done in September, 1958. Extensive bleeding was encountered and it required eight hours to perform the

operation, and 19 pints of blood were used. The right external carotid artery was tied off at this operation, and the patient had a smooth postoperative course. A few weeks later he was readmitted to the hospital with findings indicative of an intracranial spread. Further neurosurgery is contemplated at this time.

This case illustrates a rapid-growing nasofibroma which failed to respond to X-ray therapy, surgery, and testosterone. It has become malignant by position, extended intracranially, and undoubtedly will terminate fatally .

The second three cases are illustrative of malignant lesions of the nasopharynx.

Case 1. The first of these is that of W.M., a 54-year-old white male, who was referred by a neurologist. He was first seen on October 15, 1957. Two weeks before this he had noted double vision. Approximately a year before he had noticed tinnitus followed by deafness in his right ear.

Examination revealed a convergent squint of the right eye, the right drum was off color and full, and the right submaxillary gland was enlarged. Examination of the nasopharynx revealed a granulomatous lesion in the mouth of the right eustachian tube. An audiogram showed a 34 per cent loss in the right ear and a 12 per cent loss in the left. The loss was greater in the high tones. A caloric test was done and showed no response to stimulation of the right ear and a normal response of the left. Biopsy was done under general anesthesia on the following day, and the tissue was reported to show transitional cell carcinoma.

Irradiation was started, and aside from the usual skin and mucus membrane reactions his course has been uneventful. The reaction has produced a blockage of his left eustachian tube, so that he now has a bilateral deafness necessitating his wearing a hearing aid. His VIth nerve palsy has cleared up and the cervical node has disappeared. His prognosis must remain guarded for several more years.

This case is presented to show the usual intra-cranial extension which involved the VIth nerve, eustachian tube, and middle ear. This is a radiosensitive tumor which has not produced bone erosion, and so the outlook is somewhat encouraging.

Case 2. The second of the malignant cases is that of G.S., a white male 55-years-old, who was first seen on April 23, 1955. His complaint at this time was a feeling of fullness of the ears.

Examination revealed that both drums were slightly retracted. The eustachian tubes were inflated, and both were obstructed. We saw him again in April, once in May, and three times in June with this same complaint. His tubes were inflated on each occasion. He was referred to an internist in July for a complete examination which was essentially negative. He complained of pain in the cheek, and X-ray films revealed a carious tooth root, which was extracted by his dentist. Pain in the cheek persisted after the extraction, and he was admitted to Baker Hospital, where a roentgenogram on July 30, 1955, revealed a large mass in the nasopharynx. This was examined by biopsy and was reported as lymphosarcoma. X-ray therapy was begun and for a while the patient improved; however, distant metastases occurred, and in spite of further X-ray treatments and the use of nitrogen mustard, the patient died in February, 1956.

This case is illustrative of the difficulty in arriving at a correct diagnosis and the ease with which one may be misled by the commonplace

complaints of tinnitus, partial deafness and the finding of dental caries as a possible cause of pain in the face area.

Case 3. The third case is that of E.L., a 56-year-old white male who was first seen May 26, 1955. He complained of difficulty in swallowing, difficulty in raising his right shoulder, and hoarseness, all of about six weeks' duration.

Examination revealed evidence of involvement of cranial nerves IX, X, and XI (right) with partial paralysis of the right side of the soft palate, loss of sternomastoid and trapezius function, and paralysis of the right vocal cord with the right cord in the mid-line. Inspection of the nasopharynx was essentially negative.

Biopsy of the nasopharynx was done on the following day under general anesthesia and showed epidermoid carcinoma.

His local lesion was controlled with X-ray therapy and the use of radium applicators, but he died of liver metastasis on April 9, 1957.

The third malignant case shows what nerves may be involved when the tumor involves the region of the jugular foramen. It also shows the difficulty of visualizing the tumor, even when one is sure it is in the nasopharynx.

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SOME RELATIONSHIPS BETWEEN PITCH DISCRIMINATION AND SPEECH DEVELOPMENT.*

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INTRODUCTION.

Clinicians dealing with hearing-handicapped children have long been aware that such children are prone to have difficulty in achieving normal speech development. The explanation given for this observation has been that because of the hearing impairment, the child is unable to hear and differentiate the many variations in sound which make up human speech. Without a subjective awareness of these variations, the child is unable to imitate and thus produce accurately the complex combinations of sound which we call human speech; however, we are all aware that abnormal speech development and articulation problems are not limited to the hearing-handicapped child alone. In any active clinical practice or hearing and speech center many children with articulation difficulty are seen in whom no hearing impairment is evident. In attempting to postulate why these children should have such difficulty, the possibility of an alteration in the function of pitch discrimination was considered. It would seem logical that an inability to appreciate fine changes in pitch might be a factor in preventing a child from subjectively experiencing the difference between certain sounds. The inability to be aware of such differences would then limit the ability of the child to imitate accurately and thus produce human speech.

With this reasoning as a background, an attempt has been made on the basis of clinical testing to determine whether or not a significant relationship exists between the ability

*Read as a Candidate's Thesis at the Meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, New York, N. Y., Jan. 8, 1959.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 14, 1959.

to discriminate between pitches (frequencies) and the development of normal speech.

REVIEW OF THE LITERATURE.

Pitch is one of the attributes of a tone as heard by a listener and is, therefore, a subjective auditory experience.[11] Pitch is usually corollated with the frequency of sound waves as this corollation is so obvious. An individual normally will describe the pitch of a tone as low when the sound wave-length is long and will describe the pitch as progressively higher when the sound wave-length becomes progressively shorter.[7] Thus, we have the tendency to corollate pitch with frequency as a direct relationship.

Numerous investigators have shown, however, that pitch is also a function of intensity[12,15,19,21] and duration.[14,24] The variation of pitch with intensity is very small. Pollack[15] showed that the ability to discriminate between pitches improves at high intensity and becomes poorer as the intensity lessens. He also showed that as threshold is approached, an "atonal interval" is encountered where differential pitch discrimination is absent. Morgan, Garner, and Galambos[13] showed that pitch tends to drop with increasing intensity if the frequency is below 1000 cycles, but that it tends to rise if the frequency is above 4000 cycles. At 2000 cycles, there is very little variation with intensity change. Harris[10] emphasized the importance of the method used in making such determinations, and the variations which occur with the method employed. The relationship of duration to pitch is more difficult to quantitate. Turnbull[24] demonstrated that if tones are very short in duration (less than six msec.) it is impossible for most subjects to associate pitch with them. As the duration increases up to 12 to 15 msec., pitch association is possible. Once the duration exceeds this threshold, however, the pitch experience becomes only subjectively better established in the subject's consciousness. As can be seen from the preceding, although both intensity and duration are related to pitch, the major relationship is between frequency and pitch.

Shower and Biddulph[18] showed that for frequencies below

1000 cycles the just-noticeable difference in the frequency of two pure tones is about three cycles. Above 1000 cycles the number of cycles that a tone must change in order to sound noticeably different is proportional to the frequency of the tone. As the frequency of the tone increases, the size of the just-noticeable difference increases proportionately. Somewhat larger differences in frequency are necessary if the tone is less than 50 decibels above the hearing threshold. Boring[5] reviewed the procedures of previous investigators and suggested reasons for discrepancies in the DL's for frequency obtained. Rosenblith and Stevens[16] reported smaller values than Shower and Biddulph, but showed that the DL's obtained varied with the procedure used.

Wyatt[26] and Seashore[17] have both demonstrated that there is a wide variation among normal individuals in the ability to discriminate between pitches. Seashore[17] felt that this is essentially an innate ability and that such a deficiency could not be appreciably improved by practice and learning. Wyatt,[26] however, showed examples where marked improvements had been achieved in both children and adults after training in pitch perception and discrimination. Galambos[7] expressed the feeling that the pitch experience in man results from a combination of an inborn ability to analyze the difference between tonal frequency and the acquired skill in doing so. It is a function for which a neurological basis is provided, but each individual may use and cultivate it or not as he will.

Although attempts at localization of the function of pitch perception have yielded much information, many aspects of this question remain unanswered. Steinberg[20], and Bekesy and Rosenblith[2] felt that a sharply localized region of the basilar membrane was specifically excited by each frequency. Tasaki[22,23] on the basis of information gained from stimulating single auditory nerve fibers in a guinea pig, has provided data regarding the response of the auditory nerve fibers. Some respond only to low tones, some to low and middle range tones, and others to tones of all frequencies. When the high frequency limit of a fiber is reached, the sensitivity of the fiber drops abruptly and provides a characteristic cutoff point for the specific fiber. Tasaki[22,23] reasoned that this cutoff

point was perhaps analogous to the cutoff in mechanical motion of the basilar membrane reported by Bekesy[1] after observation of cochlear models. This data would tend to support the contention that low frequencies arouse activity over most of the basilar membrane and its related nerve endings, while progressively higher frequencies produce involvement of progressively smaller areas. Microelectrode studies at the level of the cochlear nucleus by Galambos, Rose and Hughes[3] have shown that a place on the basilar membrane is directly related to a place in the cochlear nucleus. Davis[4] stated that each second-order neuron is "tuned" to a particular frequency. Less acoustic energy is required at this frequency than at any other to set up impulses. With increasing intensity this "tuning" becomes less sharp, and the neuron responds to an increasingly broader band of frequencies. Studies at cortical levels have been mainly by extirpation. Evarts[6] reported that the removal of auditory cortex in monkeys producing degeneration of over 90 per cent of the medial geniculates failed to abolish pitch perception. Similar studies by Girden[9] on the dog, and by Diamond and Neff[5] on the cat have been reported. These studies would suggest that tone discrimination is still possible when the cortical termination of the auditory pathway is severely damaged.

EXPERIMENTAL DESIGN.

This study was undertaken in an attempt to determine on the basis of clinical testing whether or not in children a significant relationship exists between impaired pitch discrimination and defective speech development.

Subjects.

Children between the ages of nine and 14 years were selected. Although younger children would have been desirable from the standpoint of an increased incidence of impaired speech development, they were excluded because an exploratory study revealed marked difficulty in their accurate comprehension and performance of the experimental procedures. Only children who showed at least normal intelligence levels as determined by one of the following tests: Stanford-Binet, Form L or M, WISC, the Columbia Mental Maturity Scale,

and the International Leiter Scale were used. Multiple tests were used so that the hard-of-hearing children would not be penalized by a pure verbal test. The subjects were then divided into three categories: *1.* children with normal hearing and normal speech; *2.* children with normal hearing and articulation defects severe enough to require speech therapy; and *3.* children with impaired hearing and impaired speech development.

Equipment.

An Audivox audiometer, Model 7B, was used for pure tone testing. A Panacoustic speech audiometer, Model SA-101, was used for speech testing. A General Radio beat frequency oscillator, Model 1304-A, was used to deliver the test tones for pitch discrimination. This oscillator allowed changes in frequency to be made in one cycle increments. A Grason Stadler attenuator, Model 350, was used in order that the frequencies might be presented at 50 db above threshold. The test frequencies were delivered to the subject's ears through the PDR 10 earphones.

Materials.

The C.I.D. recordings using Spondee (W-1) and PB (W-22) words were used for speech audiometry. Basic articulation tests using pictures, words, and sentences for procuring an articulatory error profile for each child were used.

Procedures.

Pure tone audiometry, speech audiometry, and basic articulation testing was then administered to each child. These test procedures were carried out in order that each child might be placed in the proper category. Frenquency (pitch) discrimination was then carried out in the following manner: The three speech frequencies, 500, 1000, and 2000 cycles, were used for testing. The subject was instructed in the techniques of the testing, and varying changes in frequency were demonstrated to him. A practice period was permitted to insure complete familiarity with the test materials and procedures. When the examiner was satisfied that the subject understood the test procedure, actual testing was begun. With each of

the three tones, 500, 1000, and 2000 cycles, as a reference point at an intensity 50 db above threshold, the just-noticeable difference in pitch, both ascending and descending, was then determined. Five readings were obtained for each value, and the average determined. This procedure was carried out for each ear individually. Originally, the value was also determined for both ears together; however, since analysis of initial data revealed no essential change for this value, it was not obtained in later testing, and is not included in this experiment. The typical response sheet is shown in Fig. 1.

Name:..... Date:.....
Age:.....

RIGHT EAR.

D	500	A	D	1000	A	D	2000	A
1		1	1		1	1		1
2		2	2		2	2		2
3		3	3		3	3		3
4		4	4		4	4		4
5		5	5		5	5		5
Average								

LEFT EAR.

D	500	A	D	1000	A	D	2000	A
1		1	1		1	1		1
2		2	2		2	2		2
3		3	3		3	3		3
4		4	4		4	4		4
5		5	5		5	5		5
Average								

D—Descending.
A—Ascending.

FIGURE 1.

ANALYSIS AND DISCUSSION OF DATA.

This experiment was designed to compare the performance of normal, articulation defective, and impaired hearing children on frequency DL's at 500, 1000, and 2000 cycles. These frequencies were selected because of their contribution to speech intelligibility. In evaluating these performances, analysis of variance ("F" values)* provided a good method for indicating significant differences between frequency, side

*"F" value: The "F" value is a statistic to determine the variance ratio between the performance of individuals in a group and the performance of the group.

TABLE I.

Raw Scores for Eleven Normal Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

Subject	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
(1) B.S.	3	3	3	4	5	5	5	6	7	8	8	9
(2) A.M.	4	7	4	7	7	7	4	6	5	8	8	7
(3) R.C.	5	6	7	7	5	5	4	7	7	6	5	5
(4) J.B.	6	6	6	10	7	8	4	9	9	10	9	9
(5) N.C.	8	7	6	7	7	8	8	7	5	7	4	8
(6) P.S.	7	8	7	8	7	8	7	7	9	9	9	9
(7) D.S.	7	7	10	10	8	8	11	12	8	8	10	9
(8) D.H.	8	7	7	8	9	8	7	6	10	9	8	9
(9) F.R.	6	8	8	8	8	7	9	8	10	10	12	12
(10) D.S.	5	6	5	7	8	8	8	8	11	11	11	12
(11) W.S.	8	9	9	10	10	9	10	9	12	12	13	12

R = Right Ear. L = Left Ear.

TABLE II.

Raw Scores for Eleven Defective Articulation Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

Subject	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
(1) G.D.	15	19	24	29	48	39	136	148	100	104	180	232
(2) C.T.	10	17	16	38	17	17	19	18	42	36	110	85
(3) F.L.	12	13	9	8	13	13	18	14	18	19	31	20
(4) D.K.	15	16	25	30	18	20	28	30	36	44	70	69
(5) L.R.	7	9	7	7	9	15	9	14	15	15	19	20
(6) D.T.	13	8	18	14	14	17	18	21	55	45	60	55
(7) B.D.	7	17	8	8	9	10	9	9	17	13	15	18
(8) J.R.	8	8	9	10	8	9	10	12	10	11	14	17
(9) R.J.	10	9	9	8	15	14	13	12	16	17	15	16
(10) C.B.	12	10	12	9	12	12	11	11	14	15	12	14
(11) E.P.	12	15	14	15	18	19	20	21	21	22	24	25

R = Right Ear. L = Left Ear.

(left or right), and direction (ascending or descending). The study also permitted the calculation of "t" values* for analysis of the data.

Tables I, II, and III present the raw scores in frequencies for 11 normal, 11 defective articulation, and 11 impaired hearing subjects on DL for frequency at 500, 1000, and 2000 cycles. Tables IV, V, and VI indicate the mean and the variance values for 11 normal, 11 defective articulation, and 11 impaired hearing subjects on DL for frequency at 500,

*"t" value: The "t" value is a statistic which indicates whether or not differences between the means of groups are significant.

1000, and 2000 cycles. Examination of the Tables reveals that performance for the normals shows the least variation among the subjects. The Tables also reveal the greatest variation for the impaired hearing subjects with a moderate variability for the defective articulation subjects. At 1000 and 2000 cycles both the defective articulation and the impaired hearing subjects present a wider variation with the greater variation occurring at 2000 cycles. Further inspection of these Tables exhibits an increase in the mean DL's from low to high frequency.

TABLE III.

Raw Scores for Eleven Impaired Hearing Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

Subject	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
(1) M.A.	108	116	106	114	108	118	110	114	212	242	210	222
(2) E.D.	54	54	58	58	108	106	110	110	144	136	130	120
(3) B.P.	152	104	114	104	152	118	150	116	498	484	500	456
(4) S.S.	112	116	112	114	124	122	124	120	244	232	236	232
(5) R.L.	59	74	69	66	106	114	106	116	236	212	240	208
(6) R.D.	60	48	58	46	78	66	84	68	110	110	108	112
(7) C.B.	84	36	84	36	102	48	114	48	184	52	184	52
(8) B.R.	49	46	47	44	44	48	43	44	50	60	48	54
(9) D.R.	52	57	52	58	110	102	108	96	110	106	112	108
(10) S.B.	52	52	43	54	126	120	122	122	254	250	252	250
(11) J.S.	28	22	17	20	20	34	22	30	134	141	200	300

R = Right Ear. L = Left Ear.

Tables VII, VIII, and IX present the analysis of variance with respect to frequency, side and direction for the normal, articulation defective, and impaired hearing subjects. Table VII reveals an "F" value of 12.64 for performance on frequency DL's in the normal subjects. This reveals a significant difference between the performance at the different frequencies since the "F" value at the 1 per cent level of confidence is 4.78. A value of 12.64 is significant beyond the 1 per cent level and consequently, permits the rejection of the null hypothesis.* Examination of the Table reveals also that the differences between the means with respect to side

*Null hypothesis: The null hypothesis states that differences in performance among individuals or groups may be due to chance and consequently, are not real or significant differences.

TABLE IV.

Mean and Variance Values for Eleven Normal Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
X	6.09	6.73	6.55	7.82	7.36	7.36	7.00	7.73	8.45	8.91	8.82	9.18
σ^2	2.89	2.42	4.27	3.16	2.25	1.65	6.20	3.22	5.27	3.09	7.36	4.76

R = Right Ear. L = Left Ear.

TABLE V.

Mean and Variance Values for Eleven Defective Articulation Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
X	11.00	12.82	13.73	16.00	16.45	16.82	26.45	28.18	31.27	31.00	50.00	51.91
σ^2	8.20	17.16	40.42	121.20	122.27	66.36	1,354.27	1,615.56	715.82	737.60	2,818.80	4,168.49

R = Right Ear. L = Left Ear.

TABLE VI.

Mean and Variance Values for Eleven Impaired Hearing Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
X	73.64	65.91	69.09	64.91	98.00	90.55	99.36	89.45	197.82	184.09	201.82	192.18
σ^2	1,331.25	1,048.89	986.29	1,021.09	1,420.00	1,170.47	1,364.05	1,224.87	14,149.16	14,932.09	14,013.16	14,476.36

R = Right Ear. L = Left Ear.

TABLE VII.

Analysis of Variance for Eleven Normal Subjects for Frequency, Side, and Direction on DL at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

Source	SS	df	MS	F
Frequency	98.11	2	49.05	12.64*
Side	10.94	1	10.94	2.82
Direction	4.36	1	4.36	1.12
F × S	2.38	2	1.19	.31
F × D	3.32	2	1.66	.43
S × D	1.48	1	1.48	.38
F × S × D	1.11	2	.55	.14
Within	465.64	120	3.88	....
Total	587.33	131		

*	.01	.05
$F_{120\text{-}2}$	4.78	3.07
$F_{120\text{-}1}$	6.84	3.92

TABLE VIII.

Analysis of Variance for Eleven Defective Articulation Subjects for Frequency, Side, and Direction on DL at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

Source	SS	df	MS	F
Frequency	17,635.56	2	8,817.78	8.98*
Side	56.03	1	56.03	.....
Direction	4,103.76	1	4,103.76	4.18
F × S	9.38	2	4.69	.....
F × D	1,567.74	2	783.87	.....
S × D	14.67	1	14.67	.....
F × S × D	4.11	2	2.05	.....
Within	117,861.64	120	982.18	.....
Total	141,252.88	131		

*	.01	.05
$F_{120\text{-}2}$	4.78	3.07
$F_{120\text{-}1}$	6.84	3.92

and direction were not significant. Only on frequency was the DL performance statistically significant.

Table VIII reveals an "F" value of 8.98 for the defective articulation subjects on frequency performance. Since the value of 4.78 is significant at the 1 per cent level, the obtained "F" value for frequency is significant beyond the 1 per cent level and also permits the rejection of the null hypothesis. Examination of the Table also reveals an "F" value of 4.18 for

direction. Since an "F" value of 3.92 is significant at the 5 per cent level, the obtained "F" value of 4.18 is statistically significant between the 1 per cent and 5 per cent levels of confidence. These two "F" values indicate significant differences of performance for both frequency and direction. This finding of a statistically significant difference on direction reflects inferior performance on this task when compared with the normals. This inferiority may be a contributing factor to defective articulation since cues requiring precise discrimination for phonetic analysis may be lacking. Examination of the Table also reveals no significant difference in performance for side.

TABLE IX.

Analysis of Variance for Eleven Impaired Hearing Subjects for Frequency, Side, and Direction on DL at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

Source	SS	df	MS	F
Frequency	386,820.42	2	193,410.21	34.57*
Side	2,539.70	1	2,539.70	.45
Direction	42.61	1	42.61	.....
F × S	180.55	2	90.27	.....
F × D	443.68	2	221.84	.....
S × D	24.62	1	24.62	.....
F × S × D	73.04	2	36.52	.....
Within	671,377.09	120	5,594.81	.....
Total	1,061,501.72	131		

	* .01	.05
F_{120-2}	4.78	3.07
F_{120-1}	6.84	3.92

Table IX presents the "F" values for frequency, side, and direction for the impaired hearing subjects. The obtained "F" value for frequency of 34.57 is significant beyond the 1 per cent level of confidence. Inspection of Tables VII, VIII, and IX reveals that the impaired hearing subjects have the greatest differences on DL's for frequency. Examination of Tables IV, V, and VI reveals that the defective hearing subjects also exhibit the greatest difficulty in frequency discrimination. The mean values at 2000 cycles for ascending and descending performance range from 184.09 to 201.82

cycles. When compared with the normals whose performance ranged from 8.45 to 9.18 cycles, the differences are more than 20 times as great. This inability to discriminate within narrower ranges indicates that their discrimination bands are wide, which would provide a source for error. The difference in the performance of the defective articulation group while not so large as the impaired hearing group is larger than the normal group. This phenomenon is reflected in the language development of the two groups. In addition to retarded speech development the child with impaired hearing often also has a retarded language development, while the child with articulatory defects presents a retarded speech development but not a retarded language development. This point would tend to validate further the articulatory error profiles obtained on the children in these two groups. The defective hearing children consistently showed more errors than the defective articulation children, and the errors which they showed were more gross; that is, errors of distortion and substitution.

Table X presents the mean differences, degrees of freedom, and "t" values for DL for frequency at 500, 1000, and 2000 cycles for normal and defective articulation subjects. The "t" values with the exception of the performance on the descending at 1000 cycles are statistically significant. This indicates that the performances of the defective articulation subjects on every aspect except descending at 1000 cycles are inferior. These findings are consistent with the previous analysis.

Table XI presents the mean differnces, degrees of freedom, and "t" values for DL for frequency at 500, 1000, and 2000 cycles for normal and defective hearing subjects. The "t" values for very relationship are statistically significant at below the 1 per cent level of confidence. These values unequivocally indicate the inferiority of the defective hearing group on frequency DL when compared with the normal group. These findings support the previous analysis and indicate that the defective hearing subjects require a very large frequency discriminant before they can make just-noticeable difference judgments.

TABLE X.

"D" and "t" Values for Eleven Normal and Eleven Defective Articulation Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

	500 Cycles				1000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
"D"												
$X_1 - X_2$	4.91	6.09	7.18	8.18	9.09	9.45	19.45	20.45	22.82	22.09	41.18	42.73
df	20	20	20	20	20	20	20	20	20	20	20	20
"t"												
D/C	4.89	4.57	3.56	2.43	2.70	3.80	1.75	1.69	2.82	2.69	2.57	2.19

R = Right Ear. L = Left Ear. "t".01 = 2.85 "t".02 = 2.528 "t".05 = 2.086

TABLE XI.

"D" and "t" Values for Eleven Normal and Eleven Impaired Hearing Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
"D"												
$X_1 - X_2$	67.55	59.18	62.55	57.09	90.64	83.18	92.36	81.73	189.36	175.18	193.00	183.00
df	20	20	20	20	20	20	20	20	20	20	20	20
"t"												
D/C	6.13	6.05	6.59	5.92	7.97	8.06	8.28	7.73	5.28	4.75	5.41	5.04

R = Right Ear. L = Left Ear. "t".01 = 2.85 "t".02 = 2.528 "t".05 = 2.086

TABLE XII.

"D" and "t" Values for Eleven Impaired Hearing and Eleven Defective Articulation Subjects on DL, Both Ascending and Descending, for Frequency at 500, 1,000, and 2,000 Cycles for Right and Left Ears.

	500 Cycles				1,000 Cycles				2,000 Cycles			
	Ascending		Descending		Ascending		Descending		Ascending		Descending	
	R	L	R	L	R	L	R	L	R	L	R	L
"D"												
$X_1 - X_2$	62.64	53.09	55.36	48.91	81.55	73.73	72.91	61.27	166.55	153.09	151.82	140.27
df	20	20	20	20	20	20	20	20	20	20	20	20
"t"												
D/C	5.68	5.39	5.73	4.80	6.89	6.95	4.64	3.81	4.53	4.06	3.88	3.41

R = Right Ear. L = Left Ear. "t".01 = 2.85 "t".02 = 2.528 "t".05 = 2.086

Table XII presents the mean differences, degrees of freedom, and "t" values for DL for frequency at 500, 1000, and 2000 cycles for defective articulation and impaired hearing subjects. The "t" values for every relationship are statistically significant at below the 1 per cent level of confidence. The values reveal that the impaired hearing subjects are unequivocally inferior to the defective articulation subjects in DL's for frequency; thus, analysis of the data shows that while the defective articulation group is inferior in performance to the normal group, it is superior to the impaired hearing group. The impaired hearing group performance is markedly inferior to both of the other groups.

The findings throughout the study are consistent, and after verification by statistical analysis, support the hypothesis that pitch discrimination is a factor contributing to the development of speech and language for hearing impaired children; moreover, the study suggests that pitch discrimination may be a contributory factor in the development of speech for children with articulation defects.

Since it has been suggested earlier in this discussion that the functions of pitch perception and pitch discrimination are at least partially learned functions, it would seem possible that further study in this area might be indicated regarding the possible relationship to the therapy of articulation defects. It may be possible that new psychological techniques of learning can be developed for improving pitch perception and discrimination, and in this way further help with the problem of defective articulation.

SUMMARY AND CONCLUSIONS.

On the basis of clinical testing of DL for frequency of three groups of children—children with normal hearing and normal speech; children with normal hearing and articulation defects severe enough to require speech therapy; and children with impaired hearing and impaired speech development—the following conclusions were reached:

1. The normal subjects were most consistent in their performance at all frequencies, while the impaired hearing

subjects were the least consistent. The defective articulation subjects were between the other two groups.

2. All three groups exhibited a progressive increase in the mean DL for frequency from low to high frequency.

3. Statistical analysis revealed the figures obtained for performance on frequency DL's in all three groups were statistically significant. In addition, the figure obtained for direction (ascending and descending) for the defective articulation group was also significant. A possible clinical application of this observation was discussed.

4. On the basis of both raw scores and statistical analysis of the results obtained, the performances of the defective articulation subjects on DL for frequency were found to be inferior to the normal group in almost every respect.

5. On the basis of both raw scores and statistical analysis of the results obtained, the performances of the defective hearing subjects on DL for frequency were found to be markedly inferior to both of the other groups in every aspect.

The findings support the hypothesis that pitch discrimination is a contributing factor in the development of speech and language for hearing impaired children. The findings also suggest that pitch discrimination may be a contributory factor in the development of speech for children with articulatory defects. Various clinical aspects of these conclusions were discussed. The possibility of further study in the direction of improving pitch perception and pitch discrimination as an aid in improving speech development was suggested.

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FIBROUS DYSPLASIA OF THE TRACHEA AND LARYNX.*

Report of a Case.

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Fibrous dysplasia of the trachea and larynx has not been described to any extent in medical literature. It is characterized by connective tissue proliferation but can hardly be considered a true neoplasm. Because of its growth, it has the ability to cause obstruction, and for this reason behaves much like a true tumor. The possibility that it was preceded by inflammation must always be kept in mind. Occasionally, it is considered as an inflammatory hyperplasia, but I prefer the term "dysplasia," since this prefix signifies disordered growth or abnormality of development sometimes resulting in difficult or painful function of the organ involved.

CASE HISTORY.

The patient is a 54-year-old white female housewife. Her chief complaint was shortness of breath, which was increasing in severity for the past 18 months. She was referred to me for consultation by the State Tuberculosis Sanatorium on February 26, 1957, because of her shortness of breath which was becoming more pronounced, especially upon exertion. The patient had been previously diagnosed as asthma, but failed to respond to treatment. She stated that further studies in her home town revealed the presence of tubercular bacilli, and she was consequently transferred to the tuberculosis sanatorium for treatment; but the studies at the sanatorium failed to reveal the presence of tubercular bacilli in the sputum, and the radiological studies of the chest revealed a normal chest. An attempt was made to bronchoscope the patient at the sanatorium by one of the chest surgeons, and he passed the scope between the cords but could not descend beyond this point as the patient became very cyanotic and nearly died before she could be revived by means of artificial respiration and oxygen.

The patient gave no past history of severe throat infections, diphtheria, or laryngitis, nor did she ever have any severe bronchitis or pulmonary infections. There was no history of injury to the neck.

PHYSICAL EXAMINATION.

The ear, nose and throat examination was normal and non-contributory.

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*Read at the Meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., Jan. 23, 1959.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 28, 1959.

Fig. 1.

There were no palpable nodes in the neck, and X-ray of the sinuses was normal. The larynx by indirect laryngoscopy revealed a normal epiglottis, pyriform sinuses, arytenoids, as well as normal true and false vocal cords which showed good movement on respiration and phonation. Through the glottic chink could be seen an encircling type of granuloma, or cicatricial type of tissue, located at about the level of the cricoid or first tracheal ring, which had a central aperture of about 3 or 4 mm. Oral temperature was 98.4° F.; pulse was 84/minute; blood pressure 130/80 mm., and heart normal.

LABORATORY STUDIES.

X-ray studies of the neck, as reported by the radiological department, as shown on Fig. 1: "soft tissue studies of the cervical area, show a symmetrical constriction of the trachea at the level of the junction between the cricoid and the first tracheal ring. In relation to the cervical spine, the constriction point on the lateral view measures approximately 4 mm. The constriction is rather sharply localized, and the trachea appears to be of essentially normal diameter approximately 1 to 2 cm. distal to the area of narrowing." Impression: Tracheal constriction at level of cricoid and first tracheal ring.

Blood studies were normal and the serology and urinalysis negative. Sedimentation rate was 34 mm. per 60 minutes.

HOSPITAL COURSE.

On March 19, 1957, a tracheoplasty was done extending the incision from the thyroid notch to the sternal notch down to the trachea and laryngeal box. A tracheotomy tube was placed into the trachea through the sixth tracheal ring. The fifth tracheal ring was saved while the tracheal incision extended from the fourth tracheal ring through the cricoid as well as the thyroid cartilage. A sharp submucous elevator was used to dissect the hard fibrous tissue from the anterior, lateral, as well as the posterior surfaces of the first tracheal ring, cricoid, and thyroid cartilage. A skin graft was wrapped and sutured around a No. 26 French intratracheal tube to cover the denuded surface. The intratracheal tube was anchored to the tracheotomy tube below, while the upper end extended to just below the true vocal cords. Two weeks later the anchor wire from the intratracheal tube to the tracheotomy tube was cut and the intratracheal tube removed from the glottic chink, using laryngeal alligator forceps and a laryngoscope. The skin graft had taken well to the tracheal wall and remained well in place. The overlap of skin graft beyond the denuded area of the trachea sloughed off and was coughed up by the patient. The patient was discharged from the hospital April 5, 1957, or 18 days postoperative, tolerating a general diet better than before the operation, and talking fairly well in spite of the laryngeal edema that persisted.

PATHOLOGICAL EXAMINATION.

The surgical specimen was studied by Dr. Lloyd R. Hershberger[1] and reported, "The gross specimen is made up of tissue labeled as tracheal in type. It is present in 11 pieces. The largest of these pieces is 9x11x5 mm. and the remainder of the pieces are smaller in size. The smallest of the pieces is about 4 mm. in its greatest diameter. The tissue has a dense fibrous character that almost resembles cartilage. Mucosa cannot be identified grossly. The weight of this tissue is 2 grams.

"The microscopic sections (see Fig. 2) show tissue covered with squamous mucosa on one edge. The mucosa is made up of cells four or five layers in thickness and these cells are completely uniform and orderly. Occasionally one can see a transition to respiratory type of columnar epithelium. Below the mucosa, broad zones of dense fibrous connective tissue are encountered. Edema is found immediately under the mucosa, and then the deeper tissue is more dense in its character. Blood vessels (see Fig. 3) are moderately numerous and lymphocytes have infiltrated around these blood vessels. The cells (see Fig. 4) of the connective tissue are spindle in type and have remained completely uniform and orderly. Hyalinization of the connective tissue has occurred in some areas.

"Special connective tissue stains showed these fibers to be of fibrous

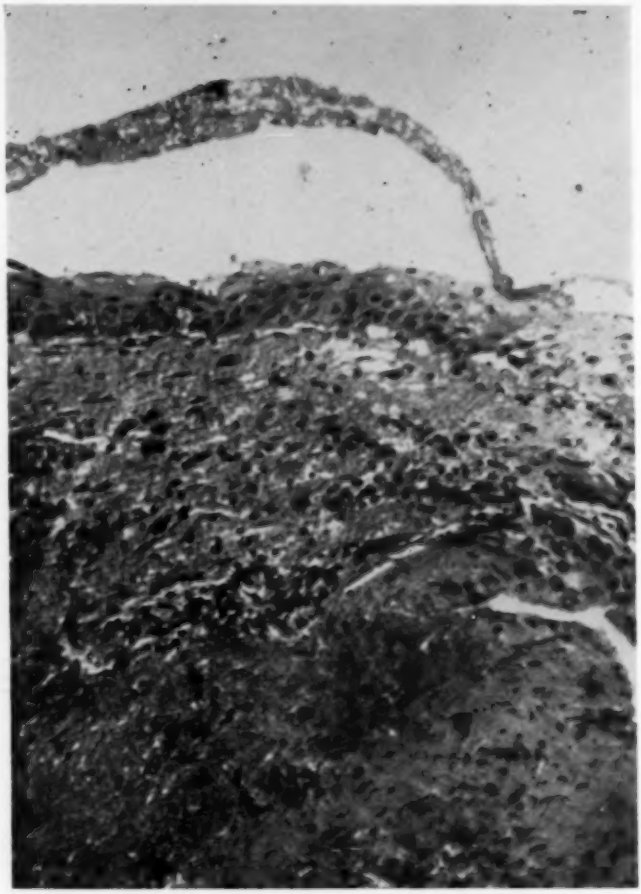

Fig. 2.

origin. The tissue shows only this fibrous reaction, and there is no evidence of a specific type of infection or malignant change.

"Diagnosis: Trachea showing a great amount of fibrosis, all benign; namely, fibrous dysplasia."

OUTPATIENT FOLLOW-UP AND DISCUSSION.

The tracheotomy tube was removed April 29, 1957. Observation a month

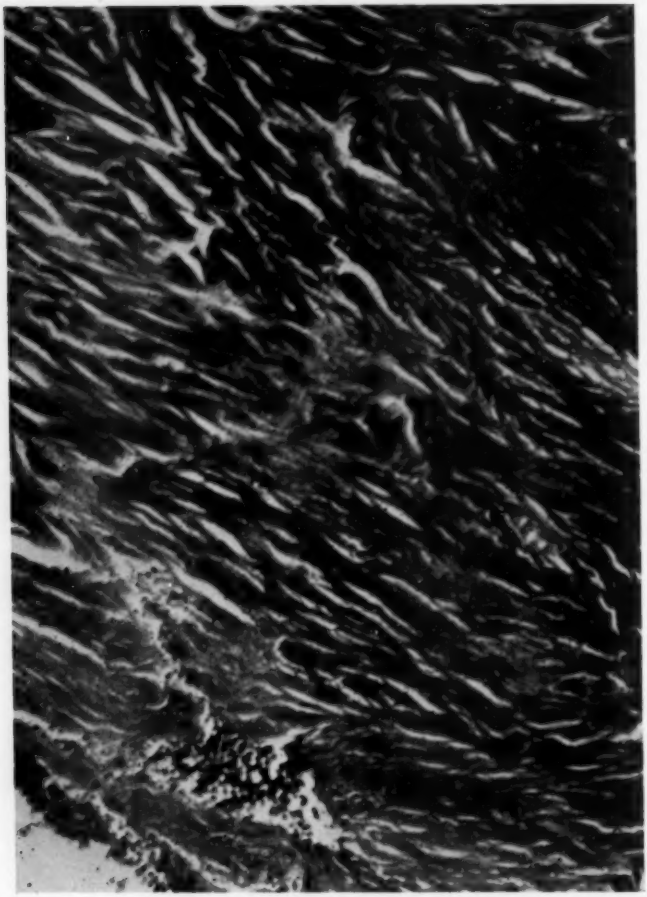

Fig. 3.

later revealed the skin graft to have taken well; but some thickening appeared to be developing in the area of the band, and she was started on hydrocortone, 25 mgm. q.i.d. for ten days and then 25 mgm. b.i.d. following studies made by Dr. Joseph V. DeSa[2] of Bombay, India, on the use of corticosteroids in the treatment of submucous fibrosis of the palate and cheek. In a personal conversation with Dr. DeSa on this case, he felt that it likewise was a collagen degeneration of the mucosa of the trachea and larynx, and that hydrocortone might be beneficial in prevent-

ing any recurrence, such as was true in his cases reported at the Sixth International Congress of Otolaryngology in 1957.

The patient was continued on hydrocortone, 25 mgm. b.i.d., until September 5, 1957, when the drug was discontinued because the aperture of the larynx and trachea was much larger, and the patient had no wheezing or shortness of breath.

In a very comprehensive report in 1932, Jackson and Jackson[3] discussed benign tumors of the trachea and bronchi in great detail and named a large number of such tumors found in their case records; but no mention is made of any case

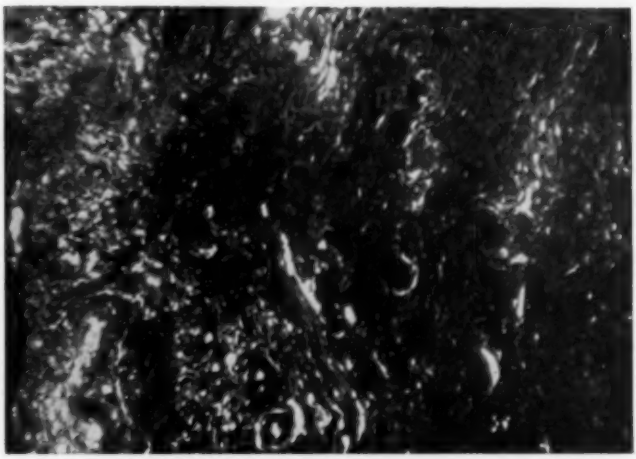

Fig. 4.

such as reported today. Jackson and Jackson state "a growth that causes death is not benign in the strictest meaning of the word, and an inflammatory hyperplasia shaped like a tumor is not, histologically, a neoplasm, but is the clinician's primary concern in tumor-like nonmalignant obstructions of the tracheobronchial tree."

It is fairly certain that if the constricting nonmalignant band in the trachea of my patient had not been removed, death from obstruction would have been inevitable, had the band continued to grow or if she had developed an acute episode of

laryngeal tracheitis unless an emergency tracheotomy had been performed.

The patient was last seen on March 19, 1958, when she reported no choking or wheezing and breathing was normal. Examination at that time revealed the aperture in the subglottic and trachea area within 80 per cent of normal or complete patency. There was very slight crusting on the left lateral wall, while the remaining area was very satisfactory.

SUMMARY.

Fibrous dysplasis is rarely encountered in the head and neck, and a review of medical literature fails to reveal any references to fibrous dysplasia of the larynx and trachea. An original case report of fibrous dysplasia of the larynx and trachea has been presented with the radiological and histological findings.

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AMERICAN HEARING SOCIETY MOVES.

National headquarters of the American Hearing Society were moved to 919 18th St., N. W., Washington 6, D. C. The agency occupies first floor space in an eight-story building which is being remodeled and converted from apartments to offices.

A METHOD OF REMOVING A NASAL HEMANGIOPERICYTOMA.*

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The purpose of this paper is to present a tumor that is of infrequent occurrence, to confirm methods of treatment recommended elsewhere and particularly to describe a method of intranasal excision.

In a recent article by Parker[1] and associates, it was shown that hemangiopericytoma may recur after 26 years. The case cited recurred following surgical removal in 1931 without subsequent X-ray therapy which is now used. In the case report which follows, X-ray therapy was administered because of the obvious angiomatous type of the tumor. This follows Dr. K. H. Kent's[2] recommendation for Roentgen therapy for this comparatively rare tumor. It is believed that this will greatly lessen the chances of recurrence.

In 1949 Stout[3] summarized the data of 25 cases of this lesion collected up to that time. Pedowitz[4] and associates collected 144 cases between 1942 and 1954. These tumors are commonly found in the soft tissues of the body and may arise from almost any site where capillary vessels are present. McCormack and Gallivan[5] reviewed 14 cases in which hemangiopericytoma was encountered at the Cleveland Clinic between 1935 and 1954. They found that the predominant complaint was that of an enlarging mass which might occur anywhere in the body. Although there have been several reports of this tumor's occurring in the orbit,[6,7] it has not been possible to find a report of its presence in the lumen of the nose. In a report by Havens and Lockhart[8] there was no specific mention of hemangiopericytoma occurring in the nose. These authors stated that the chief treatment was in interstitial irradiation.

*Read at the Meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., Jan. 23, 1959

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 28, 1959.

The pathology of this tumor is characterized by the fact that the cells are in clusters outside of the lumen of the blood vessels. This is best shown with the reticulum stain. The hemangio-endothelioma and the hemangiopericytoma can be differentiated by this microscopic difference. Here you may see a high and low power slide with reticulum stain in color. The following detailed pathological report was received from Dr. May Owen and Dr. O. J. Wollenman, Jr. Because of its interest and comparative rarity they sent their slide to the Armed Forces Institute of Pathology, where their diagnosis was confirmed. Drs. Owen and Wollenman's report:

Gross Findings: The specimen submitted, stated to be removed from the left side of the nose, consists of a mass of tissue that measured 2.5x1.7x1.3 cm. Three surfaces are covered by brownish blue, mucous membrane in which a few hemorrhages can be seen. On sectioning through this specimen, it is found to be composed of grayish white, firm and myxomatous, edematous tissue in which also scattered hemorrhages are evident. This specimen is non-friable.

Microsections of the tissue removed from the left side of the nose, reveal a very unusual tumor which is characterized by numerous vascular patterns with rather prominent endothelial cells, beyond which there is seen a uniform but highly cellular type of tumor containing an occasional mitotic figure. The cells tend to be uniform in size, having an oval nucleus with finely dispersed chromatin and an indistinct cytoplasm. With the reticulum stain, the vascular pattern is seen, and these cells appear to be outside the basement membrane, that is, to be pericytes. They are seen in small clusters in this area and in spite of the occasional mitotic figure the nuclear arrangement is uniform. There is a varying degree of collagen seen best between the vascular tumor and the mucosal surface. There is no demarcating capsule to the tumor, and histologically it is seen in the line of resection both at the base and laterally. We have studied this tumor extensively and feel that it should be classified as a hemangiopericytoma. We are somewhat disturbed by the prominence of endothelial budding seen, but we cannot identify the malignancy. With the special

stain we feel that the tumor cells are actually not endothelial but, as stated, pericytes.

These tumors are rather rare and usually behave as benign tumors for many years. There are reported cases of malignant metastasizing hemangiopericytomas and histologically it is difficult, in many instances, to differentiate those that would behave in a benign manner from those that metastasize. If not completely removed, of course, they recur, and there are reported cases in which recurrence over a period of years eventually terminated in distant metastasis. The largest collected series was reported by Dr. Arthur Purdy Stout in *Cancer*, Vol. 2, pages 1027-1054, 1949.

Pathological Findings: Hemangiopericytoma, Nasal.

The report of the Armed Forces Institute of Pathology sent to Drs. Owen and Wollenman follows:

"Members of the staff have studied the slides which you submitted in the case of Mrs. J.A.K., but are unable to agree on the best classification for this lesion. The majority agree with you that this is a vascular tumor and suggest either hemangio-endothelioma or hemangiopericytoma. The small round cells which sometimes spindle out resemble glomus cells, but sometimes they appear to line vascular spaces or have a perithelial-like arrangement. The organoid pattern, however, which one would expect to see in a glomus tumor is not a feature of this lesion; so that one would be forced to agree that it is either a hemangiopericytoma as you suggest, or a hemangio-endothelioma."

CASE REPORT.

Mrs. J.A.K. presented a complaint of bleeding from the nose on Jan. 7, 1955. On inspection of the naso-pharynx, a large, light gray tumor could be observed at the level of the base of the uvula. The tumor could also be seen in the left middle meatus of the nose, coming from the area of the natural ostium of the maxillary sinus. This tumor was extremely vascular and had the appearance of an angioma.

Method of Removal.—Because of the tremendous size of the growth and excessive bleeding it was decided to precede removal with ligation of the external carotid. This was done to prevent a disastrous hemorrhage on severance of the pedicle. On Jan. 21, 1955, under local anesthesia, a very large growth measuring 21.5x1.7x1.3 cm. was removed from the nose and naso-pharynx by pulling the soft palate forward and up, grasping the distal end of the growth with a tonsil tenaculum, and forcing a bent nasal

snare to the base of the pedicle. This was done by forcing the snare against the soft palate and pulling it forward with a tonsil hook introduced anteriorly through the inferior meatus. The pedicle was then severed. The bleeding was controlled with trombin packs. Oxycel packs were left in place at the site of removal of the pedicle. X-ray treatment was started immediately. 3000 *r* were given on each side of the nose in the area where the pedicle was severed.

On Dec. 31, 1958, Mrs. J.A.K. was free of recurrence.

CONCLUSION.

The removal of any angiomatous type of tumor should be followed with X-ray therapy to prevent recurrence.

In this case the patient was fortunate in having a hemangiopericytoma amenable to intranasal excision.

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103 East Central.

A PRACTICAL TECHNIQUE FOR INTRANASAL ETHMOIDECTOMY AND AN EVALUATION OF ITS USEFULNESS.*†

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In my experience in teaching residents at the Massachusetts Eye and Ear Infirmary the hardest surgical procedure from a practical instructional point of view is intranasal ethmoidectomy. This operation has been called by Mosher[1] "the blindest and most dangerous in all surgery." It can be made not "blind" and relatively not dangerous by a simplified but effective technique, which will be described subsequently. Every rhinologist should have the capacity to perform this operation at will and do it with whatever thoroughness is possible by the intranasal approach.

Sometimes one sees a patient who asserts that his ethmoid has been exenterated intranasally, only to find upon inspection of the field that almost all structures are intact, including the entire middle turbinate. One has the impression that occasionally a rhinologist will curette a few middle meatal anterior ethmoid cells and call this "intranasal ethmoidectomy" which, of a kind, it is; but a complete intranasal ethmoidectomy means exenteration of all cells of the labyrinth which can be reached without undue difficulty by the operator; that is, clearing of the "ethmoid box" but not of its cellular prolongations, as over the orbit. Although J. A. Pratt[2] was able to do this without sacrificing the middle turbinate, most operators remove part or all of this structure.

TECHNIQUE.

The adult patient is prepared for surgery by having a phar-

*Read at the Meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, New York, N. Y., Jan. 8, 1959.

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 15, 1959.

maseal enema the night before and 1 gm. of chloral hydrate at bedtime. Appropriate pre-operative medications one hour before operation are demerol, 50 mg., and atropine sulfate, 0.6 mg. In patients having asthma, hay fever, hives or eczema, or a family history of these conditions, it is well to avoid opium derivatives, aspirin, and barbiturates by mouth.

I prefer general anesthesia, chiefly for convenience, because the operation is often combined with several other procedures. Intratracheal gas-oxygen-ether is the usual combination of anesthetic agents, except when subepithelial diathermy coagulation cauterization of the inferior turbinates is necessary as a secondary procedure, in which case intravenous pentothal and intratracheal nitrous oxide-oxygen are used. If only one ethmoid sinus is to be exenterated without other surgery, local anesthesia is satisfactory, using 2 per cent procaine with 1/50,000 adrenalin, 1 to 2 cc. to block the nasocilliary nerve and the posterior ethmoidal nerve on the medial superior wall of the orbit near the anterior and posterior ethmoidal foramena; 1 cc. of the same solution is injected into the region of the sphenopalatine ganglion through the pterygopalatine canal. With general anesthesia, induction is made with the patient *lying down*—after which he is placed in a sitting position with care to watch the blood pressure levels when the change of position is made. With local anesthesia the patient is in a sitting position before starting the surgery.

With unconscious patients, a postnasal pack is placed in the nasopharynx and secured externally with silk strings attached to it through the mouth. About a yard of uterine tape moistened with normal saline is packed around the pharyngeal part of the intratracheal tube. The patient's face, nasal vestibules and the nasal cavity just behind each vestibule is prepared with 1/1,000 aqueous zephiran. Surgical cleanliness is employed at all times, even though the operator will be working in a non-sterile field. The purpose is not to complicate the procedure by adding organisms not already present and to proceed as though the operator actually had a sterile field. The middle of a sterile towel is draped over inch-wide adhesive and strapped firmly across the upper lip with the

tape lying at about the level of the cymba of the concha of each ear, the ends engaging each other at the occiput. A cotton cap, previously placed on the head, avoids the adhesive's sticking to the hair.

The nose is packed with a Mosher strip soaked in a 1/1,000 aqueous adrenalin wrung dry, with half the strip in each nasal cavity. Packing of the nose is done gently but blindly with a hemostat. If local anesthesia is employed, 2 per cent cocaine with 1/10,000 adrenalin is used on Dowling packs, one placed in the atrium of the nose down toward the vestibule and the second along the inferior turbinate to the posterior tip of the middle turbinate.

There are six types of mechanical problems which face the surgeon at the beginning of the operation:

1. A deviation of the septum toward the side being operated on;

2. Complete filling of the nasal cavity with polyps and obliteration of landmarks thereby;

3. A straight septum or a deviation of the septum away from the operative side with signs of infection or a few polyps, but with good visualization of the middle turbinate;

4. Turgescence of nasal mucosa in cases of vasomotor rhinitis even after shrinkage;

5. A narrow anterior and wide posterior ethmoid as defined by X-ray examination;

6. A narrow nose and narrow ethmoid with poor definition of the middle turbinates.

Correction of the deviated septum toward the side of the labyrinth to be exenterated aids a great deal in the approach to the ethmoid cells. Less time is required usually to correct such a septum than is wasted in accepting its mechanical annoyance. The septal resection is *not* limited, and is carried about 1 to 1.5 cm. higher than the inferior border of the middle turbinate. This border is about 2.5 to 3 cm. from the cribriform plate in the average nose.

If the nasal landmarks can be made out without particular difficulty, the middle turbinate is excised by cutting obliquely across it with turbinate scissors, making certain that the cut goes as high as the insertion or lamella. This usually requires closing the turbinate scissors twice instead of once in two steps from before backward. An ordinary nasal polyp snare threaded with *tonsil* wire is then engaged over the free anterior end of the middle turbinate as near as possible to the lamella, thereby excising about the anterior third or half of this turbinate. A beak-shaped middle turbinate stump remains posteriorly and is left as a landmark temporarily.

The ethmoidal bulla which always contains one posterior cell and one or two anterior cells is then entered with a Lempert zero or double-zero curette, which is swept downward.

In attempting to enter the bulla care must be taken not to confuse the medial superior roof of the antrum with the ethmoidal lateral mass, since this would cause unnecessary damage to the orbit and its contents. If the bulla is not easily identified, a flat, thin, blunt-edged septal dissector can be used to palpate very gently the region of the middle turbinate overhang in the direction of the olfactory slit without touching the cribriform plate. This in conjunction with estimating the size of the ethmoid in antero-posterior X-ray films will permit a curette to be plunged blindly into the bulla by going backward and slightly upward, but staying well away from both the roof of the antrum and the region just identified by palpation with a septal dissector as described. Once a small opening has been made into the ethmoidal bulla, the upper blade of a Greene punch (see Fig. 1) is placed inside the bulla cell with the lower jaw lying inferior to the cell; the tissue thereby engaged is evulsed. This area is then enlarged further with one or two bites of the same punch. Any polypoid tissue within the cells is stripped away. No attempt is made at this point to enter any of the *anterior* ethmoid cells not contained in the bulla.

With the same Lempert curette a puncture is made into the posterior superior portion of the bulla cell in the direction of the lowermost posterior ethmoid cells, making a common wall

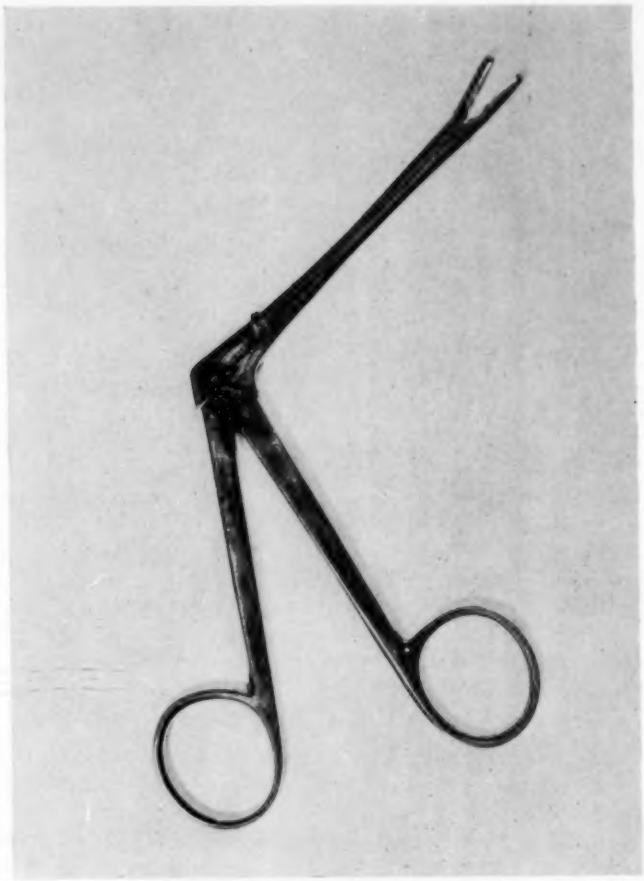

Fig. 1. Greene Punches.

with the sphenoid. The curette is again swept downward toward the floor of the nasal cavity. This maneuver will permit the small end of a Faulkner curette (see Fig. 2) to enter a large posterior ethmoid cell. The cutting edge of the ring of this instrument is kept in the frontal plane, with the flat part of the ring always parallel to the orbital plate of the frontal

Fig. 2. Faulkner Curette.

bone. The curette is pulled from behind forward, keeping *away* from the region of the cribriform plate and risking possible damage to the thin orbital wall, which rarely occurs. The resulting mass of tissue, which is pulled downward and forward into the nasal cavity, will open up the surgical field in such a manner that the operator can see a small part of the orbital plate of the frontal bone, and usually a common wall of another posterior ethmoid cell.

Since the operator is not certain as to the exact plane of this common wall in relation to the choana, he will then excise with a polyp snare, threaded with tonsil wire, all of the remainder of the middle turbinate except a small posterior stump. This small stump is left so that sphenopalatine vessels entering the nose through the foramen can be easily clamped. These vessels come in just superior to the posterior tip attachment and lie chiefly on the external side of the curved stump. They are clamped with a small, long, straight hemostat and rarely cause any serious trouble. It is a curious fact, however, that in patients who have had allergy of one variety or another, barbiturate intravenous anesthesia is sometimes associated with a *rise* in blood pressure during the operation, which may cause extra bleeding from these and other vessels. Any polyps which happen to interfere with good visualization of the posterior ethmoid already entered are excised. For this purpose I prefer a Greene punch. It is now possible to visualize the postnasal pack at the choana. This permits orientation of the operator in relation to any remaining posterior ethmoid cells lying anterior to the face of the sphenoid. These cells are then excised with particular care to avoid the lateral posterior superior relationship with the sphenoid since that is the danger point for possible damage to the optic nerve. In 171 skulls studied at the Harvard Medical School, posterior ethmoid cell dehiscences in near-contiguity to the optic nerve were found in only two. There are usually only three or four posterior cells at most, and it is easy to dissect them because of their large size. The same method described for exenteration of the bulla is utilized.

The operator is then prepared to exenterate the four remaining groups of anterior ethmoid cells[1] all at once, by plac-

ing the small ring of the Faulkner curette against the orbital plate of the frontal bone and sweeping it forward. Remembering that the instrument will lie *above* the horizontal plane of the cribriform plate, he will keep the instrument directed *away* from this structure and carry it straight forward and slightly toward the orbital side of the field. Since the instrument has an inside diameter of 5 mm. and the anterior ethmoid cells occupy about this amount of space from side to side, the cells of the frontal recess will be exenterated at the end of the sweep forward, while the downward sweep to the nasal cavity will lie against the lacrimal cells and part of the agger nasi cell. It is then a simple matter to trim away with a Greene punch the remaining anterior cells, including those around the hiatus semilunaris. This will complete the removal of the major portion of the ethmoid cells; it is then possible to use the same septal dissector as described above to palpate, with the utmost gentleness, the position of the cribriform plate and to excise a portion of the middle turbinate overhang, or to trim this structure on its lateral aspect. This trimming is done in such a manner as to remove conchal bone and approximate mucosal edges to each other. All tissue fragments and bone chips are removed as dissected by using both instruments and spot suction with various sizes of Pilling suction tubes. In many cases the ostium of the frontal sinus will be seen, or a malleable thin silver probe will enter it easily. The mucosal lining of the ostium should not be disturbed, but abnormal ethmoidal mucosa near it may be stripped away.

The superior and supreme turbinates are usually left *in situ,* but not invariably.

In an "easy" ethmoid the exenteration can be done in 20 to 30 minutes; in a difficult case the time required may be rarely an hour. If polypoid degeneration has filled the nasal cavity with polyps and made the middle turbinate unrecognizable, the polyp masses can be removed until the choana, inferior turbinate and septum are well defined. Then the exenteration can proceed as described.

It is now possible to exenterate the second ethmoid if this is

indicated. Other procedures can be easily combined with the foregoing such as: intranasal antrostomy; the making of a window into the sphenoid through the common wall of this sinus with the posterior ethmoid; excision of an hypertrophied posterior tip of the inferior turbinate ("mulberry polyp"), and subepithelial diathermy coagulation cauterization of the inferior turbinates.

It is better *not* to pack the nasal cavities postoperatively, except when submucous resection of the septum has been done simultaneously, or unless there appears to be a serious risk of hemorrhage, as judged by the degree of bleeding during the surgery. In either case sterile finger-cot packs (covering Mosher strips), lubricated with sterile vaseline, can be safely placed near the floor of each nasal cavity, rather than very high. They are secured with heavy silk mattress sutures near the ends protruding from the vestibules, tied to each other, and the ends of the silk, cut long, are taped to the dorsum of the nose.

The mouth packing is removed, together with the post-nasal pack, while simultaneously aspirating the pharynx with suction. At this stage the patient should be sufficiently conscious to gag actively. If the nose has been packed, mouth breathing is assured by use of an airway between the teeth, and close recovery-ward observation until the patient is conscious.

Nasal packs should never be placed in the nose in an unconscious patient in whom there is no pharyngeal packing, unless the operator takes precautions to prevent possible aspiration; such an accident has been known to happen with great speed.

Postoperative orders include:

1. Watch for hemorrhage;

2. Pulse and blood-pressure every 15 minutes for three hours, then frequently;

3. Elevate the head moderately;

4. Ice cap to top of head, *not* to nose;

5. Fluids and food by mouth as soon as tolerated;

6. Demerol, 50 mg. s.c. every three hours when necessary for pain. A smaller dose is given if the patient weighs less than about 150 pounds.

7. Chloral hydrate, 1 gram at bedtime if the patient is not nauseated; this dose may be lessened as stated in 6;

8. Antibiotics are not usually necessary; if given to patients with a history of asthma, hay fever, hives or eczema, the duration should be short, and an antihistamine as well as yogurt and vitamin B complex may be prescribed at the same time. Spinal fluid antibiotic, or sulfonamide concentrations, can be disregarded unless the operator suspects dural damage as possible.

Fluid replacement therapy is not a problem in most cases; a fluid intake of two liters during the first 24 hours after operation, and of three liters daily thereafter is desirable. The nasal packing, if present, is removed on the morning after operation and not replaced.

No local nasal treatment whatsoever need be undertaken in the hospital or office prior to the tenth postoperative day. Then, at the office, dried secretions can be gently removed, keeping in mind that healing epithelization occurs on the *deep* surface of crusts, and that epithelium must not be torn away upon freeing the nose of such crusts. The least local treatment possible is done during the first two weeks after operation; thereafter very little is necessary except observation and adequate treatment of any nasal polyposis. This subject is dealt with in a separate publication.[3]

EVALUATION.

1. Psychological advantages accrue from the fact that the patient is in most instances willing for the doctor to carry out whatever *intranasal* surgery he thinks necessary; yet the same patient will frequently refuse elective operations which result in a scar on the face, no matter how inconspicuous.

2. Mechanical Advantages:

a. In vasomotor rhinitis complicated by ethmoidal sinusitis, intranasal ethmoidectomy converts the frontal plane of

much of the affected nasal cavity into a quadrangle from a rough triangle, thus widening markedly the upper half of the lateral wall of the nasal air space.

b. It permits exposure of the frontal sinus ostium from below, aiding thereby in drainage if infection is present in this sinus.

c. The great majority of the ethmoid cells can be excised, and recurrent polyposis of the area can be easily controlled by excision with *aural* steel wire snares, diathermy needle cauterization or dessication when necessary.

d. Removal of the middle turbinate may be a great advantage in vasomotor rhinitis as well as in some types of sinus disease.

e. It can be combined easily with one or more other intranasal surgical procedures, including submucous resection of the septum, exenteration of the second ethmoid, intranasal antrostomy, subepithelial diathermy cauterization of the inferior turbinates, resection of an hypertrophied posterior tip of the inferior turbinate, mucous polypectomy, etc. Any or all of these procedures may be required for the relief of a chief complaint of nasal obstruction (rarely achieved by intranasal or other sinus surgery alone).

f. Although technically more difficult, and requiring greater experience it is mechanically easier than external ethmoidectomy.

3. Purulent ethmoiditis alone can usually be relieved without surgery by a combination of local treatment and appropriate antibiotics, unless a complication such as orbital abscess has occurred. Then the *external* operation is indicated.

4. The factor of allergy often requires evaluation. For example, an examination of the nose and throat in 500 unselected asthma patients, as listed in Table I, showed a high percentage of sinus involvement as well as nasal polyposis and vasomotor rhinitis.

A major purpose of this paper is to aid the rhinologist to

have instantly available a practical intranasal surgical technique for dealing with ethmoid pathology in these cases when, in the operating room, he finds this to be necessary, or a decision is made for such surgery in the pre-surgical evaluation of the patient.

The allergist should, obviously, be given the opportunity to study and treat any patient having vasomotor rhinitis, asthma or allied conditions. Since his results in vasomotor rhinitis and nasal polyposis are often poor,[3] he will frequently request aid from the rhinologist, or the patient will insist upon

TABLE I.

Nose and Throat Lesions in 500 Unselected Asthma Patients.

Lesion	Cases	Extrinsic	Intrinsic	Miscellaneous
Pansinusitis (unilateral or bilateral)	46	11	34	1
Sinusitis—one or more (unilateral or bilateral)	316	136	180	0
Vasomotor rhinitis	223	111	109	3
Polypi	211	81	128	2
Surgically deviated septum	95	47	46	2

a rhinological opinion. The allergists themselves are divided in regard to the effectiveness of any form of surgical treatment of the sinuses[4,5] in asthma and allied conditions. The rhinologist should keep in mind the following points:

1. If prolonged steroid therapy has been part of the medical treatment, any surgery may entail grave risk of lengthy collapse of blood pressure during or within hours after operation because of an inadequate alarm reaction. The word "prolonged" may be defined as at least six months of doses substantially more than 35 mg. of Cortisone (or equivalent doses of other steroids) daily. Three months or less of such treatment can usually be disregarded, but beyond this the surgeon should take proper precautions to protect the patient from the risk of severe hypotension of several hours' duration. He can do this by refusing any surgery or by ordering the following:

a. Cortisone, 50 to 200 mg by mouth the night before operation.

b. Cortisone, 50 to 200 mg. intramuscularly two hours before operation.

c. About two hours before operation 100 mg. of hydrocortisone in 500 cc. of 5 per cent or 10 per cent glucose intravenously is started and continued as a drip for about eight hours. This drip is repeated in the same dosage when the first one is completed, unless the patient is able to take the same amount of hydrocortisone by mouth in divided doses over an eight-hour period.

d. The patient is then returned to whatever dose of steroid he was taking prior to entering the hospital.

2. In patients who have not had any steroid therapy, it is likely that the normal "alarm" reaction to any type of major operation will release enough Cortisone physiologically beyond the normal body requirements of about 35 mg. daily to result in temporary improvement in allergy manifestations in many cases. It is only in recent years that otolaryngologists and other surgeons have recognized this fact.

3. Because of the multiplicity of nasal lesions usually present, intranasal ethmoidectomy is ordinarily only one of several procedures carried out in surgical treatment, which may include the Caldwell-Luc antrum and measures required for good mechanical and physiological functioning of the nasal cavities.[6]

4. In patients having vasomotor rhinitis without asthma, about 18 per cent will develop asthma sooner or later[6] with or without any nasal operation.

5. After sinus surgery, improvement in the asthma is likely to occur in about 50 per cent of the cases with a slightly higher favorable response if the patient is followed for many years.[7] Since some asthmatics improve spontaneously, and many forms of medical treatment relieve asthma, the rhinologist should be cautious in giving his operation credit even in very favorable end results. In my own personal experience, where 105 asthma cases had intranasal ethmoidectomy, together with other nasal surgery at the same time,[7] 56 patients (55 per cent) were improved or cured of asthma over a follow-up period of six

months to 20 years. Only 12 per cent (13 cases) became worse later, in contrast to an overall expectancy of about 20 per cent for worsening by any method of tratment. In 26 external ethmoidectomies, including 11 external frontal sinus operations, the *asthma* improved in 46 per cent and was worse in 27 per cent.

6. The local result in the nose will be found satisfactory following intranasal surgery in about 75 per cent of the patients,[6] providing that in the follow-up period recurrent polypoid degenerative changes are dealt with properly.[3]

7. It is best to have the patient understand that any nasal surgery is done for the purpose of improving the local condition of the nose, and that it is not done to "cure" asthma, although it is common experience that sometimes the asthma will improve following such surgery.

SUMMARY.

1. Intranasal ethmoidectomy is difficult for residents being trained in rhinology to learn.

2. A practical method which is described in this paper may aid the beginner or expedite expert technique.

3. The usefulness of the procedure is evaluated in terms of everyday problems presented in ordinary rhinological practice.

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INTRANASAL ETHMOIDAL SCHWANNOMA.

Report of Two Cases.

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College Park, Ga.

Unusual bleeding from any orifice of the human body should make one suspicious of malignancy. This is especially true when one encounters epistaxis. While most frequently seen in trauma about the nose, hypertension, ulceration and hemangioma of the septum, acute upper respiratory infection, and hereditary hemorrhagic teleangiectasia or Rendu-Osler's disease, nosebleed is often the first sign of the presence of an intranasal tumor. It is rather unusual for a nerve tumor to cause nasal bleeding, and for this reason as well as the rare etiology, we report the following two cases:

Case 1. Mrs. M. W., a 31-year-old housewife, first noted nasal hemorrhage in October of 1949 "after taking gas" presumably for tooth extraction. Again in January of 1950 after a blow to the left side of the nose epistaxis ensued, and the nose was cauterized in the office of her local physician. X-rays of the sinuses one week prior to admission to the Massachusetts Eye and Ear Infirmary on April 20, 1950, revealed a probable cyst of the left ethmoid region. Past history revealed that during the Winter of 1943, 1944, she had allergic rhinitis that subsided following removal of nasal polypi. She did not remember which side had been involved. A biopsy apparently was not done nor the excised tissue examined under the microscope.

Under intravenous pentothal anesthesia a left intranasal ethmoidectomy was performed. A mass involving the left ethmoidal labyrinth and partly attached to the septum by adhesions was removed with a wire snare and ring punch. The specimen was cauliflower-like and had a volume of about ten cubic centimeters. Diagnosis by Dr. Tracy Mallory was: "Neurofibrosarcoma. Areas of recent hemorrhage with reaction and fibrosis. An occasional mitosis is seen" (see Fig. 1).

At last correspondence with her in September, 1958, she wrote that she was not at all well, and an appointment was made for an examination.

*Deceased, Sept. 30, 1958.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Dec. 21, 1958.

More recently the term *malignant Schwannoma* has been preferred to that of neurogenic sarcoma or even neurofibrosarcoma. Because of the unusual pathological diagnosis we put the case on the shelf for a while, hoping to find another one in clinical practice. Time went by, and we forgot about it until the Spring of 1958, when one of us (M.L.) had a referred case from South Georgia, of a firm, intranasal bleeding mass. The literature was thoroughly combed, using the Quarterly *Index Medicus* and the AF reference list from

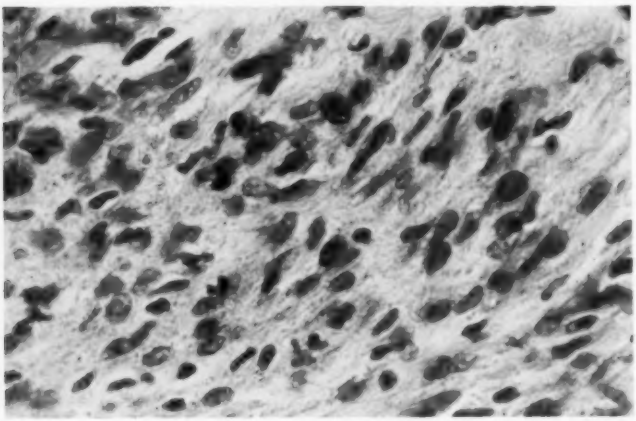

Fig. 1. Oil immersion view of tumor in Case 1, showing the pleomorphism that suggested a diagnosis of neurofibrosarcoma. Re-examination eight years later changed this opinion to that of Schwannoma, a benign tumor, malignant "by position" only.

1948 through May, 1958, and not a single tumor of such description could be found in the world literature.

Case 2. Mr. C. W., a 28-year-old sawmill operator, was examined and found to have a mass in the right ethmoid region associated with obstruction to breathing on that side for a period of about three months' duration. X-rays revealed an opaque mass in the right ethmoid region without breakdown of bone. A biopsy was taken and reported as a benign Schwannoma (see Fig. 2).

Operation at this time was refused by the patient in spite of the "malignant by position" aspect and the danger of erosion of bone and extension of infection to the meninges as a possible sequel.

Because of the similarity of the two cases re-examination

of the slides from Case 1 was made and the original diagnosis of *neuroflibrosarcoma* was changed to merely *Schwannoma*. One pathologist mentioned the fact that from the microscopic picture alone, it was difficult to differentiate between the two, and that the only way to be sure was whether the patient was alive or not after a few years; another stressed the incompleteness of our knowledge of nerve tumors in 1950 and that these cases illustrated how opinion and further knowledge and study of these unusual tumors can change over the

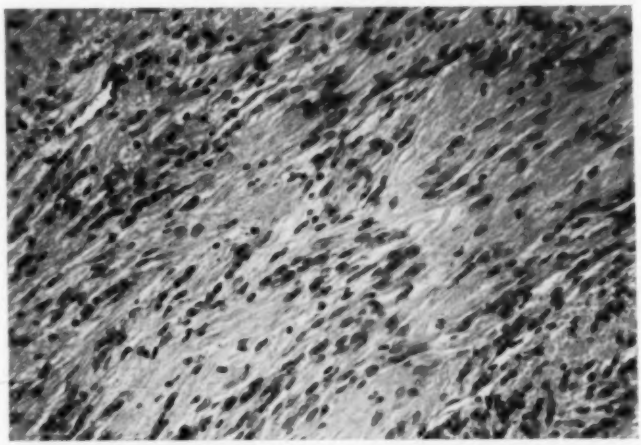

Fig. 2. High power magnification of tumor in Case 2, showing the palisading effect which is characteristic more of a benign lesion. Pleomorphism shows up well here also.

years. We just didn't know enough about the development nor the various histological types in 1950 to be as accurate as we are now.

The literature review did reveal a case of malignant Schwannoma of the tip of the nose[1] following intensive X-ray therapy for eczema. Ash and Raum[2] mentioned a fibrosarcoma of the maxillary sinus, and a fibrosarcoma of the ethmoid was noted by Stratton[3] in 1957. Endonasal Schwannoma was discussed in 1951 by Gonzalez Loza and Rosenzvit.[4]

SUMMARY.

These two cases of ethmoidal Schwannoma are illustrative of an unusual type of intranasal nerve tumor first manifesting by the rather common symptom of epistaxis. They further prove that not all intranasal masses are hemangiomata, granulomas or mere polyps. It is to be emphasized that removal of obstructive bleeding masses and examination under the microscope is the only way to be sure of our pre-operative diagnosis.

REFERENCES.

1. Conley, John J.: Malignant Schwannoma of Tip of Nose. *Arch. Otolaryngol.*, 62:638-640, Dec., 1955.

2. Ash, J. E., and Raum, Muriel: *Atlas of Otolaryngic Pathol., A.F.I.P., Am. Regis. of Path. and A. A. of O. & O.*, p. 208-209, 1956.

3. Stratton, H. J. M.: Case, Fibrosarcoma of the Ethmoid. *Jour. Laryngol. and Otol.*, 67:631-634, Oct., 1957.

4. Gonzalez Loza, M., and Rosenzvit, E.: Endonasal Schwannoma. *Ann. Otol., Rhinol. and Laryngol.*, 60::988-991, 1951.

124 West Princeton Ave.

CANADIAN MEDICAL ASSOCIATION, BRITISH COLUMBIA DIVISION, OTO-OPHTHALMOLOGICAL CONFERENCE 1959.

The 1959 British Columbia Oto-Ophthalmological Conference will be held in Vancouver, B. C., on May 28th to 30th, 1959. Guest speakers will be Dr. Alfred Huber, Assistant to Professor Amsler, Zurich, Switzerland, in Ophthalmology, and Dr. Theo. E. Walsh of St. Louis, Mo., in Otolaryngology. All Otolaryngologists and Ophthalmologists in the Pacific Northwestern area are cordially invited to attend.

For further details write: H. B. Lockhart, M.D., Chairman, 1541 West Broadway Ave., Vancouver 9, B. C.

PROGRAM FOR THE 108TH ANNUAL MEETING AMERICAN MEDICAL ASSOCIATION

Section on Laryngology, Otology and Rhinology.

Atlantic City, June 8-12, 1959.

(Rather than assigned discussers, each paper, except those of the Guest of Honor and the Chairman, will be followed by an open discussion period; questions and comments from the floor are encouraged.)

Tuesday, June 9—2 P.M.

PROF. DR. H. H. NAUMANN, Würzburg, Germany—
Intravital Observations of the Nasal Mucous Memberane (Film).

GEORGE M. COATES, M.D., Philadelphia, Pa.—
Address of Guest of Honor.

GORDON D. HOOPLE, M.D., Syracuse, N. Y.—
Modern Otolaryngology.

F. W. DAVISON, M.D., Danville, Pa.—
Laryngeal Obstruction in Children.

BEN T. WITHERS, M.D., Houston, Tex.—
Facility in T&A Management: Conclusions from 2400 Consecutive Cases.

JOSEPH H. OGURA, M.D., St. Louis, Mo.—
Cancer of the Pharynx, Larynx and Upper Esophagus; Surgical Aspects.

BUSINESS MEETING:

Report of Section Delegate to AMA House of Delegates—
Gordon F. Harkness, M.D., Davenport, Ia.
Dean M. Lierle, M.D., Iowa City, Ia., Alternate.

Report of Section Representatives to Board of Governors of American College of Surgeons—
Roderick Macdonald, M.D., Rock Hill, S. C.
Edley H. Jones, M.D., Vicksburg, Miss.
John J. Conley, M.D., New York, N. Y.

Report of Nominating Committee—
F. W. Davison, M.D., Danville, Pa.
Louis E. Silcox, M.D., Philadelphia, Pa.
John D. Singleton, M.D., Dallas, Tex.

Wednesday, June 10—2 P.M.

J. W. HAMPSEY, M.D., Pittsburgh, Pa.—
Current Concepts of Etiology and Management in Otolaryngic Allergy.

VICTOR R. ALFARO, M.D., Washington, D. C.
Address of Section Chairman.

HENRY S. KAPLAN, M.D., San Francisco, Calif.—
New Horizons in the Radiotherapy of Malignant Disease.

JOHN A. KIRCHNER, New Haven, Conn.—
Facial Bone Injuries.

M. R. HIMALSTEIN, M.D., Bay Pines, Fla.—
Obliterative Frontal Sinus Surgery Using Gelfoam.

J. M. RAVID, M.D., New York, N. Y.—
Malignant Melanoma of the Nose and Paranasal Sinuses, and Juvenile Melanoma of the Nose.

Wednesday Evening, June 10—6:30 P.M.

Social Hour and Dinner Honoring the Guest of Honor of the Section, Dr. George M. Coates.

Thursday, June 11—2 P.M.

JOSEPH L. GOLDMAN, M.D., and HARRY ROSENWASSER, M.D., New York, N. Y.—
Current Concepts of the Management of Otitic Infections.

MERLE LAWRENCE, Ph.D., Ann Arbor, Mich.—
Ear Physiology.

EDWARD W. HARRIS, M.D., Columbus, O.—
Symptoms Referable to the Eustachian Tube.

GEORGE KELEMEN, M.D., Boston, Mass.—
Maternal Diabetes and Congenital Deafness.

JOSEPH SATALOFF, M.D., Philadelphia, Pa.—
Stapes Mobilization with Long-Standing Otosclerosis.

HOWARD P. HOUSE, M.D., Los Angeles, Calif.—
Polyethylene in Middle Ear Surgery.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

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Annual Meeting: October 10, 1959, Belmont Hotel, Chicago, Ill.

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Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting:

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President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

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Meeting: Jointly with the N. C. Society of Eye, Ear, Nose and Throat. Next joint annual meeting of The South Carolina Society of Ophthalmology and Otolaryngology and the N. C. Eye, Ear, Nose and Throat Society at Charleston, S. C., Sept. 14, 15 and 16, 1959.

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Books and monographs received are acknowledged in this column. This notice may be regarded as a return courtesy to the publisher or author. Reviews will be published later as the editors may elect.

Notice of each book or monograph, including title, name of author, publisher, pagination, price, etc., will be presented in these notices so that our readers may have all data at their disposal for further inquiry.

AUDIOLOGY: PRINCIPLES AND PRACTICE. By Hayes A. Newby, Stanford University School of Melicine. 342 pages and numerous illustrations. Appleton-Century-Crofts, Inc., New York. 1958.

ORAL HISTOPATHOLOGY: A MANUAL FOR STUDENTS AND PRACTITIONERS OF DENTISTRY. By Martin A. Rushton, M.A., M.D., Odont.D., F.D.S,R.C,S, England and Edin- M,A,, M,D,, Odont,D., F.D.S.R.C.S. England and Edinburgh, Professor of Dental Medicine, University of London—and Brian E. D. Cooke, M.R.C.S., L.R.C.P., F.D.S.R. England, Reader in Dental Medicine, University of London. 190 pages with Index and 214 illustrations. The Williams & Wilkins Company, Baltimore, Md. 1959. Price $7.00.

VOICE OF THE DEAF—A BIOGRAPHY OF EDWARD MINER GALLAUDET. By Maxine Tull Boatner, Director of Historical research, Conferen,e of Executives of American Schools for the Deaf, with Introduction by John S. Brubacher. 190 pages with Index and several illustrations. Public Affairs Press, Washington, D. C. 1959. Price $4.50.

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